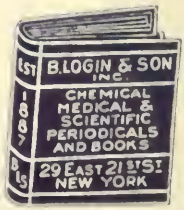
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TRANSACTIONS

of the

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Fifty-Fourth Annual Meeting

NEW LONDON, CONN., 1918

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1918

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OFFICERS AND COUNCIL

of the

American Ophthalmological Society

Elected at the Annual Meeting, July 9, 1918

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DR. LUCIEN HOWE, Buffalo, N. Y.

Vice-President

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Secretary-Treasurer

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PRESIDENTS OF THE SOCIETY

(Since its Foundation)

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1869–1873 Dr. Henry W. Williams, Boston, Mass.
1874–1878 Dr. C. R. Agnew, New York.
1879–1884 Dr. Henry D. Noyes, New York.
1885–1889 Dr. William F. Norris, Philadelphia.
1890–1893 Dr. Hasket Derby, Boston, Mass.
1894–1898 Dr. George C. Harlan, Philadelphia.
1899–1902 Dr. O. F. Wadsworth, Boston, Mass.
1903–1905 Dr. Charles S. Bull, New York.
1906 Dr. Arthur Mathewson, Washington, D. C.
1907 Dr. Charles J. Kipp, Newark, N. J.
1908 Dr. Samuel D. Risley, Philadelphia.
1909 Dr. S. B. St. John, Hartford, Conn.
1910 Dr. Samuel Theobald, Baltimore, Md.
1911 Dr. Emil Gruening, New York.
1912 Dr. Edward Jackson, Denver, Col.
1913 Dr. Myles Standish, Boston, Mass.
1914 Dr. Robert Sattler, Cincinnati, O.
1915 Dr. M. H. Post, St. Louis, Mo.
1916 Dr. George E. de Schweinitz, Philadelphia.
1917 Dr. Peter A. Callan, New York.
1918 Dr. William H. Wilder, Chicago, Ill.

American Ophthalmological Society.

FOUNDED 1864.

LIST OF MEMBERS.

* Associate Members. O. M. Original Members.

Elected.	Name.	Residence.	Place.
1896	Dr. A. E. Adams,	Balmville, Cor. North,	Newburg, N. Y.
1915	*Dr. Ellice M. Alger,	40 East 41st Street,	New York, N. Y.
1899	Dr. A. N. Alling,	257 Church Street,	New Haven, Conn.
1896	Dr. G. W. Allyn,	Empire Building,	Pittsburgh, Pa.
1882	Dr. A. Alt,	5614 Waterman Ave.,	St. Louis, Mo.
1883	Dr. J. A. Andrews,	1229 State Street,	Santa Barbara, Cal.
1913	Dr. Fred. D. Bailey,	260 Hancock Street,	Brooklyn, N. Y.
1911	Dr. Arthur J. Bedell,	344 State Street,	Albany, N. Y.
1908	Dr. Geo. H. Bell,	40 East 41st Street,	New York, N. Y.
1905	*Dr. Jas. H. Bell,	Moore Building,	San Antonio, Texas.
1916	*Dr. Eugene M. Blake,	55 Trumbull Street,	New Haven, Conn.
1909	Dr. James Bordley, Jr.,	330 N. Charles Street,	Baltimore, Md.
1881	Dr. H. W. Bradford,		Wolfboro, N. H.
1918	*Dr. H. H. Briggs,	73 Haywood Street,	Asheville, N. C.
1908	Dr. E. V. L. Brown,	122 S. Michigan Blvd.,	Chicago, Ill.
1907	Dr. Wm. E. Bruner,	Guardian Bldg.,	Cleveland, Ohio.
1901	Dr. H. D. Bruns,	211 Camp Street,	New Orleans, La.
1906	Dr. W*. K. Butler,	1207 M Street, N. W.,	Washington, D. C.
1906	Dr. W. Gordon M. Byers,	346 Mountain Street,	Montreal, Canada.
1918	*Dr. E. Phinizy Calhoun,	Candler Building,	Atlanta, Ga.
1909	Dr. L. W. Callan,	452 Fifth Avenue,	New York, N. Y.
1876	Dr. Peter A. Callan,	452 Fifth Avenue,	New York, N. Y.
1885	Dr. F. P. Capron,	118 Angell Street,	Providence, R. I.
O. M.	Dr. W. H. Carmalt,	261 St. Ronan Street,	New Haven, Conn.
1898	Dr. J. T. Carpenter,	2039 Chestnut Street,	Philadelphia, Pa.
1911	Dr. James J. Carroll,	405 N. Charles Street,	Baltimore, Md.
1904	Dr. Burton Chance,	255 So. 13th Street,	Philadelphia, Pa.
1893	Dr. H. B. Chandler,	34½ Beacon Street,	Boston, Mass.
1904	Dr. J. W. Charles,	509 Humboldt Bldg.,	St. Louis, Mo.
1893	Dr. F. E. Cheney,	64 Commonw'th Ave.,	Boston, Mass.
1898	Dr. J. H. Claiborne,	8 West 40th Street,	New York, N. Y.

Elected.	Name.	Residence.	Place.
1912	Dr. E. W. Clap,	390 Commonw'th Ave.,	Boston, Mass.
1891	Dr. C. F. Clark,	188 East State Street,	Columbus, Ohio.
1912	*Dr. G. S. Crampton,	1700 Walnut Street,	Philadelphia, Pa
1890	Dr. C. M. Culver,	36 Eagle Street,	Albany, N. Y.
1906	Dr. R. J. Curdy,	1134 Rialto Building,	Kansas City, Mo.
1902	Dr. Colman W. Cutler,	24 East 48th Street,	New York, N. Y.
1907	Dr. A. E. Davis,	47 East 57th Street,	New York, N. Y.
1878	Dr. W. S. Dennett,	8 East 49th Street,	New York, N. Y.
1918	*Dr. David N. Dennis,	221 West 9th Street,	Erie, Pa.
1909	Dr. George S. Derby,	7 Hereford Street,	Boston, Mass.
1874	Dr. Lewis S. Dixon,	232 Clarendon Street,	Boston, Mass.
1912	Dr. Oscar Dodd,	30 N. Michigan Blvd.,	Chicago, Ill.
1902	Dr. Alexander Duane,	139 E. 37th Street,	New York, N. Y.
1908	Dr. J. L. Duncan,	Jenkins Bldg.,	Pittsburgh, Pa.
1912	Dr. E. C. Ellett,	Exchange Building,	Memphis, Tenn.
1887	Dr. J. B. Emerson,	40 East 41st Street,	New York, N. Y.
1890	Dr. Arthur E. Ewing,	Metropolitan Bldg.,	St. Louis, Mo.
1896	Dr. T. H. Fenton,	1319 Spruce Street,	Philadelphia, Pa.
1915	*Dr. Carl Fisher,	102 S. Franklin Street,	Rochester, Minn.
1893	Dr. George F. Fiske,	25 E. Washington Street,	Chicago, Ill.
1909	Dr. M. L. Foster,	211 Centre Avenue,	New Rochelle, N.Y.
1904	Dr. W. H. Fox,	1826 Jefferson Place,	Washington, D. C.
1913	*Dr. Lee M. Francis,	636 Delaware Avenue,	Buffalo, N. Y.
1902	Dr. Percy Fridenberg,	38 West 59th Street,	New York, N. Y.
1894	Dr. H. Friedenwald,	1029 Madison Ave.,	Baltimore, Md.
1913	*Dr. H. G. Goldberg,	1925 Chestnut Street,	Philadelphia, Pa.
1895	Dr. G. M. Gould,	215 Atlantic Avenue,	Atlantic City, N. J.
1896	Dr. H. Y. Grant,	Falls View,	Ontario, Canada.
1908	Dr. L. S. Greene,	1624 I Street, N. W.,	Washington, D. C.
1905	Dr. Allen Greenwood,	101 Newbury Street,	Boston, Mass.
1914	*Dr. J. Milton Griscom,	1925 Chestnut St.,	Philadelphia, Pa.
1892	Dr. G. W. Hale,	210 Hitchcock Building,	Nashville, Tenn.
1895	Dr. Wm. D. Hall,	416 Marlborough Street,	Boston, Mass.
1887	Dr. H. F. Hansell,	17th and Walnut Streets,	Philadelphia, Pa.
1918	*Dr. D. F. Harbridge,	Goodrich Building,	Phoenix, Ariz.
1918	*Dr. Wm. F. Hardy,	Metropolitan Bldg.,	St. Louis, Mo.
1901	Dr. Herbert Harlan,	516 Cathedral Street,	Baltimore, Md.
1889	Dr. David Harrower,	13 Elm Street,	Worcester, Mass.
1896	Dr. N. D. Harvey,	114 Waterman Street,	Providence, R. I.
1904	Dr. H. H. Haskell,	29 Commonwealth Ave.,	Boston, Mass.
1914	Dr. Edw. B. Heckel,	Jenkins Building,	Pittsburgh, Pa.
1913	*Dr. Chas. R. Heed,	1205 Spruce Street,	Philadelphia, Pa.
1896	Dr. Ward A. Holden,	8 East 54th Street,	New York, N. Y.
1909	Dr. T. B. Holloway,	1819 Chestnut Street,	Philadelphia, Pa.
1906	Dr. C. R. Holmes,	8 East 8th Street,	Cincinnati, Ohio.

Elected.	Name.	Residence.	Place.
1883	Dr. E. E. Holt,	723 Congress Street,	Portland, Me.
1917	*Dr. Harvey Howard,	233 Charles Street,	Boston, Mass.
1878	Dr. Lucien Howe,	520 Delaware Avenue,	Buffalo, N. Y.
1891	Dr. D. W. Hunter,	80 West 40th Street,	New York, N. Y.
1898	Dr. J. W. Ingalls,	874 Lafayette Avenue,	Brooklyn, N. Y.
1895	Dr. E. E. Jack,	215 Beacon Street,	Boston, Mass.
1885	Dr. Edward Jackson,	318 Majestic Building,	Denver, Col.
1912	Dr. P. C. Jameson,	139 Montague Street,	Brooklyn, N. Y.
1918	*Dr. J. Wilkinson Jervey,		Greenville, S. C.
1892	Dr. W. B. Johnson,	170 Broadway,	Paterson, N. J.
1908	Dr. B. R. Kennon,	Taylor Building,	Norfolk, Va.
1901	Dr. H. W. Kilburn,	192 Marlborough Street,	Boston, Mass.
1903	Dr. Arnold Knapp,	10 East 54th Street,	New York, N. Y.
1889	Dr. Carl Koller,	681 Madison Avenue,	New York, N. Y.
1888	Dr. Chas. W. Kollock,	86 Wentworth Street,	Charleston, S. C.
1913	Dr. Robert S. Lamb,	Stoneleigh Court,	Washington, D. C.
1891	Dr. W. E. Lambert,	112 East 35th Street,	New York, N. Y.
1902	Dr. W. B. Lancaster,	522 Commonw'lth Ave.,	Boston, Mass.
1912	Dr. H. M. Langdon,	2018 Chestnut Street,	Philadelphia, Pa.
1903	Dr. J. C. Lester,	616 Madison Avenue,	New York, N. Y.
1907	Dr. George F. Libby,	Metropolitan Bldg.,	Denver, Col.
1883	Dr. J. A. Lippincott,	Jenkins Building,	Pittsburgh, Pa.
1878	Dr. F. B. Loring,	1420 K Street,	Washington, D. C.
1909	Dr. R. G. Loring,	10 Arlington Street,	Boston, Mass.
1899	Dr. D. B. Lovell,	State Mutual Bldg.,	Worcester, Mass.
1914	*Dr. W. H. Lowell,	101 Newbury Street,	Boston, Mass.
1918	*Dr. Hunter H. McGuire,	227 West Water Street,	Winchester, Va.
1913	Dr. A. L. Macleish,	1101 Brockman Bldg.,	Los Angeles, Cal.
1891	Dr. F. W. Marlow,	731 University Block,	Syracuse, N. Y.
1878	Dr. W. V. Marmion,	1211 13th Street, N. W.,	Washington, D. C.
1868	Dr. Arthur Mathewson,	Cosmos Club,	Washington, D. C.
1905	Dr. S. C. Maxson,	235 Genesee Street,	Utica, N. Y.
1907	Dr. C. H. May,	698 Madison Avenue,	New York, N. Y.
1874	Dr. C. S. Merrill,	27 S. Hawk Street,	Albany, N. Y.
1908	Dr. H. S. Miles,	417 State Street,	Bridgeport, Conn.
1883	Dr. J. Lancelot Minor,	Bank of Commerce Bldg.,	Memphis, Tenn.
1881	Dr. W. F. Mittendorf,	399 Park Avenue,	New York, N. Y.
1916	*Dr. John H. Ohly,	22 Schermerhorn St.,	Brooklyn, N. Y.
1887	Dr. H. S. Oppenheimer,	276 Madison Avenue,	New York, N. Y.
1908	Dr. W. R. Parker,	David Whitney Bldg.,	Detroit, Mich.
1908	Dr. S. McA. Payne,	542 Fifth Avenue,	New York, N. Y.
1895	Dr. R. J. Phillips,	123 So. 39th Street,	Philadelphia, Pa.
1869	Dr. Thomas R. Pooley,	26 Trinity Street,	Newton, N. J.
1898	Dr. W. Campbell Posey,	2049 Chestnut Street,	Philadelphia, Pa.
1895	Dr. H. R. Price,	435 Clinton Avenue,	Brooklyn, N. Y.

Elected.	Name.	Residence.	Place.
1906	Dr. Brown Pusey,	7 West Madison Street,	Chicago, Ill.
1902	Dr. Walter L. Pyle,	1931 Chestnut Street,	Philadelphia, Pa.
1901	Dr. Alex. Quackenboss,	143 Newbury Street,	Boston, Mass.
1906	Dr. McCluney Radcliffe,	1906 Chestnut Street,	Philadelphia, Pa.
1885	Dr. Alex'r Randall,	1717 Locust Street,	Philadelphia, Pa.
1892	Dr. R. L. Randolph,	609 Park Avenue,	Baltimore, Md.
1891	Dr. J. M. Ray,	Atherton Bldg.,	Louisville, Ky.
1901	Dr. R. G. Reese,	50 West 52nd Street,	New York, N. Y.
1889	Dr. R. A. Reeve,	48 Bloor Street East,	Toronto, Canada.
1899	Dr. H. O. Reik,	506 Cathedral Street,	Baltimore, Md.
1912	Dr. W. G. Reynolds,	1165 Dean Street,	Brooklyn, N. Y.
1884	Dr. Stephen O. Richey,	732 17th Street,	Washington, D. C.
1901	Dr. G. O. Ring,	17th and Walnut Streets,	Philadelphia, Pa.
1895	Dr. H. W. Ring,	185 Church Street,	New Haven, Conn.
1908	Dr. J. Norman Risley,	36 7th Street,	New Bedford, Mass.
1880	Dr. Samuel D. Risley,	2018 Chestnut Street,	Philadelphia, Pa.
1900	Dr. Wm. K. Rogers,	188 East State Street,	Columbus, Ohio.
1899	Dr. Dunbar Roy,	Gr. Opera House Blk.,	Atlanta, Ga.
1875	Dr. John D. Rushmore,	129 Montague Street,	Brooklyn, N. Y.
1880	Dr. Robert Sattler,	30, The Groton,	Cincinnati, Ohio.
1912	Dr. A. C. Sautter,	1421 Locust Street,	Philadelphia, Pa.
1898	Dr. T. B. Schneideman,	1831 Chestnut Street,	Philadelphia, Pa.
1889	Dr. G. E. de Schweinitz,	1705 Walnut Street,	Philadelphia, Pa.
1895	Dr. P. N. K. Schwenk,	1417 North Broad St.,	Philadelphia, Pa.
1918	*Dr. Wm. E. Shahan,	Metropolitan Bldg.,	St. Louis, Mo.
1913	Dr. C. E. G. Shannon,	1633 Spruce Street,	Philadelphia, Pa.
1905	Dr. John R. Shannon,	17 East 38th Street,	New York, N. Y.
1916	*Dr. Francis W. Shine,	24 East 48th Street,	New York, N. Y.
1911	Dr. J. F. Shoemaker,	1006 Carleton Bldg.,	St. Louis, Mo.
1902	Dr. Wm. A. Shoemaker,	1006 Carleton Bldg.,	St. Louis, Mo.
1902	Dr. Wm. T. Shoemaker,	109 South 20th Street,	Philadelphia, Pa.
1905	Dr. E. A. Shumway,	2046 Chestnut Street,	Philadelphia, Pa.
1918	*Dr. Dorland Smith,	834 Myrtle Avenue,	Bridgeport, Conn.
1912	Dr. E. T. Smith,	Conn. Mutual Bldg.,	Hartford, Conn.
1910	Dr. Albert C. Snell,	53 S. Fitzhugh St.,	Rochester, N. Y.
1909	Dr. F. M. Spalding,	39 Huntington Ave.,	Boston, Mass.
1878	Dr. J. A. Spalding,	627 Congress Street,	Portland, Me.
1911	*Dr. Chas. N. Spratt,	900 Nicollet Avenue,	Minneapolis, Minn.
1884	Dr. Myles Standish,	72 St. James Avenue,	Boston, Mass.
1873	Dr. George T. Stevens,	40 East 41st Street,	New York, N. Y.
1903	Dr. Edw. Stieren,	Union Arcade Bldg.,	Pittsburgh, Pa.
1887	Dr. T. Y. Sutphen,	992 Broad Street,	Newark, N. J.
1900	Dr. Wm. M. Sweet,	1205 Spruce Street,	Philadelphia, Pa.
1917	*Dr. William Tarun,	605 Park Ave.,	Baltimore, Md.

Elected.	Name.	Residence.	Place.
1888	Dr. Lewis H. Taylor,	83 So. Franklin Street,	Wilkesbarre, Pa.
1880	Dr. Samuel Theobald,	Cathedral & How'd Sts.,	Baltimore, Md.
1893	Dr. Ch. Hermon Thomas,	3634 Chestnut Street,	Philadelphia, Pa.
1903	Dr. Edgar S. Thomson,	405 Park Avenue,	New York, N. Y.
1898	Dr. Jas. Thorington,	2031 Chestnut Street,	Philadelphia, Pa.
1908	Dr. Andrew Timberman,	112 East Broad Street,	Columbus, Ohio.
1911	Dr. Fred. T. Tooke,	368 Mountain Street,	Montreal, Canada.
1912	*Dr. E. Török,	8 East 54th Street,	New York, N. Y.
1906	Dr. H. H. Tyson,	11 East 48th Street,	New York, N. Y.
1916	*Dr. Derrick T. Vail,	24 East 8th Street,	Cincinnati, Ohio.
1898	Dr. C. A. Veasey,	Paulsen Building,	Spokane, Wash.
1905	Dr. F. H. Verhoeff,	101 Newbury Street,	Boston, Mass.
1903	Dr. F. L. Waite,	68 Pratt Street,	Hartford, Conn.
1916	*Dr. Clifford B. Walker,	721 Huntington Avenue,	Boston, Mass.
1873	Dr. David Webster,	24 East 48th Street,	New York, N. Y.
1893	Dr. J. E. Weeks,	46 East 57th Street,	New York, N. Y.
1914	Dr. Walter B. Weidler,	137 East 60th Street,	New York, N. Y.
1900	Dr. C. D. Wescott,	22 E. Washington Street,	Chicago, Ill.
1916	*Dr. John M. Wheeler,	80 West 40th Street,	New York, N. Y.
1891	Dr. Edward Wheelock,	26 Gibbs Street,	Rochester, N. Y.
1892	Dr. J. A. White,	200 E. Franklin Street,	Richmond, Va.
1895	Dr. W. H. Wilder,	1417 Peoples Gas Bldg.,	Chicago, Ill.
1910	Dr. Carl Williams,	School L. & Greene St.,	Philadelphia, Pa.
1912	Dr. Edw. R. Williams,	1069 Boylston Street,	Boston, Mass.
1893	Dr. Wm. H. Wilmer,	1610 I Street, N. W.,	Washington, D. C.
1906	Dr. J. Scott Wood,	172 Sixth Avenue,	Brooklyn, N. Y.
1900	Dr. Hiram Woods,	842 Park Avenue,	Baltimore, Md.
1915	*Dr. H. W. Wootton,	319 Lexington Avenue,	New York, N. Y.
1881	Dr. J. P. Worrell,	20 South 7th Street,	Terre Haute, Ind.
1905	Dr. Wm. Zentmayer,	1506 Spruce Street,	Philadelphia, Pa.
1903	Dr. S. Lewis Ziegler,	1625 Walnut Street,	Philadelphia, Pa.

Total 196

HONORARY MEMBERS.

1868	Dr. J. S. Prout,	Fishkill,	Dutchess Co., N.Y.
O. M.	Dr. F. P. Sprague,	229 Commonw'lth Ave.,	Boston, Mass.

Whole number 198

IN MEMORIAM

DR. WILLIAM G. CRAIG, elected 1903
DR. NEIL J. HEPBURN, elected 1890
DR. HARRIS G. SHERMAN, elected 1906
DR. CHARLES H. WILLIAMS, elected 1874
DR. F. M. WILSON, elected 1888

Dr. Charles H. Williams

NECROLOGY.

CHARLES H. WILLIAMS, A.M., M.D.

MAJOR WALTER B. LANCASTER,
M. R. C., U. S. Army.

Dr. Charles H. Williams died June 9, 1918, of valvular heart disease. His health had been failing for a long time. He joined this Society in 1874, perhaps the youngest member ever admitted, as he was only twenty-four years old at the time, having been born April 19, 1850.

His father was the first professor of ophthalmology at Harvard, one of the founders of this Society, and, indeed, of American ophthalmology.

Dr. Williams was born in Boston. He graduated from Harvard in 1871. His favorite sport in college was rowing. After receiving his A.B., he spent a year in post-graduate work at Harvard and at the Massachusetts Institute of Technology, giving special attention to physics. He was made Master of Arts by Harvard in 1872.

He entered the Harvard Medical School, and after receiving his degree of M.D., spent two years in special study in Europe under very favorable auspices. Horner of Zurich, Jaeger of Vienna, Snellen and Donders of Holland, Landolt of Paris, Bowman and others at Moorfields, were the teachers who influenced him most.

Returning to Boston, he began the practice of ophthalmology, being associated with his father. He did some teaching and a good deal of hospital work. In 1886 he

accepted a position as ophthalmic surgeon on the Chicago, Burlington & Quincy Railroad, where he had much to do with the development of the Burlington Protective System.

In 1895 Dr. Williams came back to Boston and resumed private practice. He did not take on any hospital clinics, but did some important work as consultant for the N. Y., N. H. & H. R. R., the Boston & Maine Railroad, and the Boston Elevated. For example, the selection of a yellow light as a cautionary signal, which was first adopted on the N. Y., N. H. & H. system, was made with his advice after practical study on the railroad at night, riding in the engine cab. This has been adopted by most of the railroads of this country and also in Germany and Australia.

Dr. Williams was an enthusiastic and painstaking worker, very accurate and careful in his office and operative work. His exceptional grasp of physiologic optics is easily understood when we consider his natural gift for mechanics, his special training in physics at Harvard and the Institute of Technology, and his studies with men like Donders, Snellen, Landolt, and the others mentioned above. He was particularly ingenious in devising and skilful in constructing apparatus of various kinds. He did important work on form sense, color sense, and light sense. Thus, his test-charts for both far and near vision are among the best. Probably no reading tests are more carefully printed than those he prepared for this Society. The electro plates are in the custody of our secretary. His work on color sense was particularly good. His office equipment for the scientific investigation and measurement of defects of color vision surpassed that of any clinic or medical school with which I am acquainted. He devised a simple pocket instrument for the rough and ready measurement of illumination. His apparatus for measuring heterophoria and cyclotropia is unsurpassed. Some day it will be given the place it deserves when suitable commercial methods give it the pub-

licity it merits and place it within our reach.* His lantern for testing the color vision of railway and other employees has often been imitated but never surpassed.

Dr. Williams married Miss Fisher, of Brookline, and has left a son and a daughter. It was a source of great satisfaction to him that both his son and son-in-law were in active service in France in the Field Artillery.

Among the societies of which Dr. Williams was a member may be mentioned:

Massachusetts Medical Society.
American Medical Association.
New England Ophthalmological Society.
American Ophthalmological Society.
Chicago Ophthalmological Society.
American Academy of Arts and Sciences.
Illuminating Engineering Society.

Dr. Williams was often asked to serve on committees, where his special knowledge and his tireless industry made him a most useful member. In his professional relations he maintained a very high ethical standard.

The following partial list of papers gives a good idea of the direction his labors took:

"Report of the Committee of the Am. Ophth. Society on Standards and Methods of Examining the Acuteness of Vision, Color Sense and Hearing, for Railway and Marine Service," 1901.

"Standards of Form and Color Vision Required in Railway Service," Trans. Am. Ophth. Society, 1897.

"Progress toward Greater Uniformity in the Examination of the Vision, Color Sense and Hearing of Railway Employees," Trans. Am. Ophth. Soc., 1902.

"The Cortical Fusion of Some Color Sensations," Trans. Am. Ophth. Soc., 1904.

"Test-Types According to the Geometrical Progression of Dr. John Green," Jour. Am. Med. Ass'n, Oct. 7, 1905.

"Report of Committee on Standard Illumination for Visual Test-Types," Trans. Section on Ophth., Am. Med. Ass'n, 1908.

"Observation on the Effect of the Light of the Mercury-Vapor Lamp on the Eye," Electrical World, Sept. 2, 1911.

"The Best Tests for Defective Color Sense," Chicago Med. Recorder, May, 1895.

* Made by Andrew J. Lloyd Co., Boston, Mass.

"Color Testing," Boston Med. and Surg. Journal, Jan. 2, 1896.
"Test-Types for Examining the Vision of Railway Employees," Jour. of the Boston Society of Medical Sciences, Mar., 1899.
"The Need of a Supplementary Lantern Test for the Proper Examination of Color Perception," Boston Med. and Surg. Jour., July 30, 1903.
"A More Uniform Standard for the Illumination of Visual Test-Types," Trans. Section on Ophth., Amer. Med. Ass'n, 1906.
"Improved Lantern for Testing the Color-Perception," Trans. Am. Ophth., 1909.
"Report of the Committee on Standards and Methods of Examining the Color Vision," Trans. Section on Ophth., Am. Med. Ass'n, 1916.

Dr. Frederick Morse Wilson.

DR. FREDERICK MORSE WILSON.

H. S. MILES, M.D.,
Bridgeport, Conn.

Dr. Frederick Morse Wilson was born at Hebron, Maine, December 8, 1850, son of the Rev. and Mrs. Adam Wilson.

Dr. Wilson graduated from Colby in 1871, and from Harvard Medical School in 1875. After two years of general practice at Waterville, Maine, he went to New York and took up the study of eye and ear disorders with Drs. Agnew and St. John Roosa.

He was Assistant Surgeon to Dr. J. B. Emerson at the Manhattan Eye and Ear Hospital for more than fifteen years.

In 1879 he settled in Bridgeport for the practice of his specialties, and here he resided until the time of his death, which occurred at Panama on December 30, 1917.

He was visiting the Isthmus with Mrs. Wilson for the purpose of attending the wedding of their younger daughter, Helen, to Mr. James R. Murray, British Consul at Colon, which event had been celebrated two weeks previous to his death. Their elder daughter is Mrs. Frank Lyman, of Montreal, Canada.

It had been necessary for Dr. Wilson to take the best possible care of himself for many years because of an existing heart lesion. A third attack of gallstone colic proved fatal.

Dr. Wilson was Ophthalmic and Aural Surgeon to the Bridgeport Hospital from its beginning until about five years ago, when he was made consultant. He was President of the Corporation at the time of his death.

Dr. Wilson's success was due, principally, to his skill as an operator. His touch was extremely light and steady. His judgment when emergencies arose was excellent.

Originally left handed, he had trained himself to use both hands equally well. He did the so-called simple cataract extraction mostly with the Snellen conjunctival flap.

He was a member of the local and state medical associations and the New York Ophthalmological Society.

Dr. Wilson was made a member of the American Ophthalmological Society in 1888. He was a regular attendant, contributing several valuable papers and often taking part in the discussions.

His hobby was the water, and he was a sailor. He progressed from canoe to catamaran and then to auxiliary yawl. It was his custom, during the early years of his membership, to sail his yacht "Duplex" from Bridgeport to New London at the time of the annual meetings, then usually held at the old Pequot House. Many of the members have partaken of his hospitality, and enjoyed short trips upon his boat after the closing of the scientific sessions.

He was one of the organizers of the Bridgeport Yacht Club and its first commodore.

During the last dozen years he had traveled widely, visiting Europe several times, Iceland, India, Australia, and South America.

Three or four months of each year were spent away from his office on these extensive vacations, but at all other times he was to be found in his office. When not attending to his practice, the time was spent in reading.

He was naturally most reserved with his patients, but with favorites and friends was quite the opposite. Most of our members will recall with pleasure the many enjoyable social hours spent with him.

MINUTES OF THE PROCEEDINGS.

FIFTY-FOURTH ANNUAL MEETING.

The Griswold,

New London, Conn., July 9, 1918.

The Fifty-fourth Annual Meeting of the Society was called to order at 9 A. M. by the President, Dr. Wm. H. Wilder, of Chicago.

The following papers were read and discussed:

1. "Solid Edema of the Face. Report of Three Cases." Walter Baer Weidler, M.D.

Discussed by Dr. Ziegler.

2. "Histological Findings in Eyeballs Removed After Unsuccessful Sclerocorneal Trephinings." W. Gordon M. Byers, M.D.

Discussed by Drs. Verhoeff, Pyle, and Jackson.

3. "Tuberculous Iridokeratitis—Some Features in Its Pathology." Fred. T. Tooke, M.D.

Discussed by Drs. Verhoeff and Weeks.

4. "Cataract in Retinitis Pigmentosa; Its Pathology and Treatment." Arnold Knapp, M.D.

Discussed by Drs. Pyle, Harlan, Knapp, and Wilmer.

5. "Microscopic Findings in a Case of Coralliform Cataract." F. H. Verhoeff, M.D.

6. "Permanent Vascular Changes Following Injuries to the Eye." Edward Jackson, M.D.

7. "A Case of Evulsion of the Optic Nerve." Eugene M. Blake, M.D.

8. "Ocular Affections Dependent Upon Disease of the Tonsils. Report of Two Cases." Howard F. Hansell, M.D.

9. "Acute Retrobulbar Neuritis of Septic Origin, Resulting in Rapid and Complete Loss of Vision." Walter Eyre Lambert, M.D.

Papers of Drs. Hansell and Lambert discussed by Drs. Shumway, J. F. Shoemaker, Weeks, G. Oram Ring, Sutphen, Hiram Woods, Pyle, Carpenter, Tarun, Byers, Wilder, and Lambert.

10. "Concerning the Etiology of Hereditary Optic Nerve Atrophy. Report of Two Cases With Interesting *X*-ray Findings." William Zentmayer, M.D.

11. "Tuberculin in Diseases of the Eye." John E. Weeks, M.D.

Discussed by Drs. Risley, Davis, Hiram Woods, Byers, Jackson, Ziegler, Verhoeff, and Weeks.

The following were invited to be guests of the Society and take part in the discussion: Capt. C. Berens, Jr., U. S. Army; Drs. M. Feingold, New Orleans, La.; C. E. Ferree, Bryn Mawr, Pa.; J. Brown Farrior, Tampa, Fla.; B. F. Young, Knoxville, Tenn.; and B. P. Croft, Greenfield, Mass.

EXECUTIVE SESSION, JULY 9TH, 9 O'CLOCK.

The meeting was called to order by the President, Dr. Wilder, who announced the following appointment:

Auditing Committee: Dr. Fred. T. Tooke.

The reading of the Minutes of the last meeting was dispensed with, as the proceedings had been published in the Transactions.

The report of the Secretary-Treasurer was read. The Auditing Committee certified to its correctness, and it was accepted and ordered filed.

The Secretary announced the following deaths during the year: Dr. Wm. G. Craig, Dr. Neil J. Hepburn, Dr. H. G. Sherman, Dr. Charles H. Williams, and Dr. F. M. Wilson.

Dr. W. R. Parker, Chairman of the Committee on Theses, reported that twelve theses had been received by the Committee, and nine were favorably recommended to the Council.

Dr. William Zentmayer, Chairman of the Committee on Standardization of the Illumination of Test Cards and Perimeters, stated that it had been impossible to hold a meeting of the Committee during the year. At his request the Committee was continued.

Dr. Edward Jackson, Chairman of the American Board for Ophthalmic Examinations, presented the following annual report of the Board:

Since the last meeting of this Society examinations have been held at New York City immediately after the meeting of the American Medical Association in June, and in Pittsburgh in October, 1917, preceding the meeting of the American Academy of Ophthalmology and Oto-Laryngology, and in Chicago on June 10, 1918. At these examinations the Certificate of the Board has been granted to 85 candidates. The printing and presenting of these certificates have been greatly delayed both on account of war service of the members of the Board who were charged with their preparation, and on account of the difficulty in getting executed engraving of the highest character for such certificates. But certificates are now being prepared, and will shortly be furnished to those entitled to them.

In spite of the fact that nearly all the members of the Board have been engaged in war service, five of them holding commissions in the army and navy, it has been possible to obtain a quorum at each meeting of the Board. This, however, has been at the cost of some hardship and sacrifice on the part of the members; and the failure of one representative of this Society to attend any of the meetings. Under present conditions it is especially important that every member of the Board should share in its labors and become familiar with its workings. It is suggested that any one chosen who cannot meet this requirement should resign his position on the Board; and that failure to take part in the meetings of the Board within one year after election

should be considered a resignation of the office, and that a successor should be chosen.

The recognition of this Board as the Credentials Committee on Ophthalmology of the American College of Surgeons brings essential coöperation in two movements largely directed to the same purpose—the raising of the general professional standards for those recognized as specialists in ophthalmic practice. With the resignation of Dr. Frank C. Todd as Secretary of the Board, he having been commissioned Lieutenant-Colonel in the National Army, and expecting orders to go to France at the head of his hospital unit, the Secretary's office was moved to the offices of the American College of Surgeons, 1123–1125 East Washington Street, Chicago, Ill., and Dr. William H. Wilder was chosen Secretary.

It is now our painful duty to announce the death of Dr. Frank C. Todd on the 4th of July at the Presbyterian Hospital in Chicago, whither he had been taken suffering from double pneumonia contracted while on duty on inspection service. Dr. Todd's work as Secretary of the Board was done faithfully and intelligently, and the records of his office were turned over in excellent shape to his successor only a few days ago.

The work of the Board has brought out the fact that the most serious defects in the training for ophthalmic practice in this country are in regard to the fundamental facts of ocular anatomy, pathology, and physiologic optics. It is hoped that the attention of the members of the Society will be directed to this matter, and that they will actively cooperate in bringing about a better recognition of what such training ought to be.

Upon motion the report was approved and ordered filed.

The report of the Council was read by Dr. Arnold Knapp, Acting Chairman, in the absence of Dr. Alexander Quackenboss in military service.

The Council announced that it had received the resignation of Dr. William M. Sweet as Secretary-Treasurer, and regretted that it must recommend that it be accepted. The

following nominations were offered for officers of the Society for the ensuing year:

President: Dr. Lucien Howe, Buffalo, N. Y.

Vice-President: Dr. Hiram Woods, Baltimore, Md.

Secretary-Treasurer: Dr. T. B. Holloway, Philadelphia.

It was moved and seconded that the Secretary cast a ballot for the nominees, which motion was carried, and the officers were declared elected.

Upon motion of Dr. John R. Shannon a vote of thanks was extended to the retiring Secretary-Treasurer for the valuable and efficient services he had rendered the Society.

The Council recommended the following named candidates for admission to the Society, and they were declared elected Associate Members:

Dr. H. H. Briggs, Asheville, N. C.

Dr. F. Phinizy Calhoun, Atlanta, Ga.

Dr. David N. Dennis, Erie, Pa.

Dr. D. F. Harbridge, Phoenix, Arizona.

Dr. Wm. F. Hardy, St. Louis, Mo.

Dr. J. W. Jervey, Greenville, S. C.

Dr. Hunter H. McGuire, Winchester, Va.

Dr. W. E. Shahan, St. Louis, Mo.

Dr. Dorland Smith, Bridgeport, Conn.

In referring the names of other candidates to the Council for 1919, attention was called to the inability of several of the candidates to prepare a thesis owing to Government service, and it was therefore recommended that that part of Section 3, Article III, of the Constitution, which applies to the time limit of two years in which a candidate must present a thesis, be waived in the case of those in the military, naval, or civil service of the Government, also that the provision of Article VI, Section 3, concerning attendance, be also waived for members of the Society in the Government service.

The Council recommended Dr. Myles Standish for elec-

tion to the American Board for Ophthalmic Examinations to serve for three years.

The Council confirmed the nomination of Dr. Walter B. Lancaster to prepare a memorial notice of the late Dr. Charles H. Williams, and of Dr. H. S. Miles to prepare a memoir of the late Dr. F. M. Wilson.

The Council nominated the following members of the Society to serve on the Board of Governors of the American College of Surgeons: Drs. Myles Standish, G. E. de Schweinitz, and Charles W. Kollock.

The Council recommended that the assessment for 1918 be $10.00.

The Council made no recommendation as to the time and place of the next meeting of the Society, as the meeting would be held in connection with the Congress of American Physicians and Surgeons, of which the Society is a component part.

Upon motion the report of the Council with the recommendations made was adopted.

In reference to the next meeting of the Congress of American Physicians and Surgeons, Dr. Carmalt reported that the Chairman of the Executive Committee of the Congress, Dr. Shands, had been unable to arrange matters with the hotels in Washington for the meeting next year, and that in all probability the Executive Committee would decide to hold the meeting in Atlantic City near the time of the meeting of the American Medical Association, so that members from the West and South would be enabled to attend the meetings of both societies while in the East.

[Since the meeting of the Society the Program Committee of the Congress has set June 16 and 17, 1919, as the dates, and Atlantic City, N. J., as the place, for the next session of the Congress. There will be no afternoon sessions of the component societies on these dates.]

The President announced the following Committees:

Council: Drs. Arnold Knapp, Wm. Zentmayer, C. F. Clark, Cassius D. Wescott, and Walter B. Lancaster.

Committee on Theses: Drs. Walter R. Parker, W. H. Wilmer, and H. F. Hansell.

Program Committee: Drs. W. T. Shoemaker, William M. Sweet, and the Secretary.

Publication Committee: Drs. Walter E. Lambert, William M. Sweet, and the Secretary.

Standardization of Illumination of Test Cards and Perimeters: Drs. Wm. Zentmayer, Geo. S. Crampton, and H. Maxwell Langdon.

Representatives on the American Board for Ophthalmic Examinations: Dr. Myles Standish, to serve for three years, Dr. Wm. Campbell Posey, to serve for two years, and Dr. J. E. Weeks, to serve for one year.

Committee on Arrangements of the Congress of American Physicians and Surgeons: Dr. William M. Sweet, Delegate; Dr. Edward A. Shumway, Alternate.

Dr. Pyle made the suggestion that the Program Committee arrange for a symposium on focal infections as related to ophthalmic conditions for the next meeting of the Society.

Dr. Howe offered a number of suggestions designed to increase the scientific value of the work of the Society. Tables were shown indicating the geographic distribution of the membership, the regularity of attendance, and the members who had contributed to the scientific programs during a long period of years.

A motion was made and carried that the recommendations of Dr. Howe be submitted to a committee of five, to report at the next meeting of the Society, and that this report shall be sent to each member as soon as completed, so that the members will have time to consider the report before the meeting at which it is to be presented.

The Chair announced the appointment of the following

committee to consider the suggestions of Dr. Howe: Drs. Hiram Woods, G. Oram Ring, Walter E. Lambert, Edward Jackson, and F. H. Verhoeff.

A motion was made and carried that the dues of any member in the Government service would be remitted upon request during the period of such service.

Dr. Hiram Woods moved that the question of incorporation of the Society be referred to the Council for consideration. Upon motion of Dr. Carmalt the question was laid upon the table.

MORNING SESSION, JULY 10TH, 9 O'CLOCK.

The Society was called to order by the President, Dr. Wilder.

Dr. Walter L. Pyle, Philadelphia, exhibited a "Trial Frame for Test Lenses, with Demonstration of a New Model."

The following papers were read and discussed:

12. "Some Ocular Conditions Affecting the Efficiency of the Aviator." W. H. Wilmer, M.D., and C. Berens, M.D.

Discussed by Drs. Duane and Wilmer.

13. "An Improved System of Illumination for the Electric Ophthalmoscope." Carl Koller, M.D.

Discussed by Drs. Lambert, Pyle, and Koller.

14. "On the Construction of Test Objects, Especially Those for Measuring Fatigue of Accommodation." Lucien Howe, M.D.

15. "The Inertia of Adjustment of the Eye for Clear Seeing at Different Distances." C. E. Ferree, Ph.D., and G. Rand, Ph.D. (by invitation).

Discussed by Drs. Howe, Duane, and Ferree.

16. "Some Observations on the Restoration of the Orbital Socket." P. N. K. Schwenk, M.D., and Wm. Campbell Posey, M.D.

Discussed by Drs. Tarun, Zentmayer, Heckel, Reeve, and Wilder.

17. "Report of Four Cases of Orbital Tumors Removed, with Preservation of Vision, Through an Orbital Incision Along the External Orbital Rim," Wm. Campbell Posey, M.D.

18. "Report of Two Orbital Tumors." A. Edward Davis, M.D.

19. "Tumor of the Orbit, With Extension to Frontal and Temporal Regions, Treated by *X*-Rays. William M. Sweet, M.D.

Discussion of papers of Drs. Posey, Davis, and Sweet by Drs. Claiborne, Reeve, Posey, and Davis.

20. "Ophthalmoscopic Conditions Simulating Sarcoma of the Choroid." Harry Friedenwald, M.D.

Discussed by Drs. Hiram Woods, Knapp, and Friedenwald.

21. "Report of Case of Transient Left Homonymous Hemianopsia." Henry H. Tyson, M.D.

Discussed by Drs. Claiborne and Wilder.

22. "A Practical Point in the Technic of Corneal Tattooing, the Value of Which is Not Commonly Recognized." Samuel Theobald, M.D.

23. "Hypermetropia Responsible for Heterophoria, Astigmatism, and Myopia." S. M. Payne, M.D.

Discussed by Dr. Claiborne.

24. "Conservative Treatment of Asthenopia in Children." Edward R. Williams, M.D.

Discussed by Dr. Payne.

Adjourned.

The following members were present and registered at the Fifty-fourth Annual Meeting:

Alger, Ellice M.
Alling, A. N.
Blake, Eugene M.
Bruner, Wm. Evans
Byers, W. Gordon M.
Callan, Lewis White
Callan, Peter A.
Capron, F. P.

Carmalt, W. H.
Carpenter, John T.
Claiborne, J. H.
Clap, Edmund W.
Cutler, Colman W.
Davis, A. Edward
Dodd, Oscar
Duane, Alexander
Foster, Matthias L.
Friedenwald, Harry
Griscom, J. Milton
Hall, W. D.
Hansell, Howard F.
Harlan, Herbert
Haskell, H. H.
Heckel, Edward B.
Heed, Chas. R.
Holden, Ward A.
Howard, Harvey J.
Howe, Lucien
Hunter, D. W.
Ingalls, James W.
Jack, Edwin E.
Jackson, Edward
Jameson, P. Chalmers
Knapp, Arnold
Koller, Carl
Lamb, Robert Scott
Lambert, W. E.
Lancaster, W. B.
Lester, John C.
Lovell, D. B.
Lowell, W. Holbrook
Manson, S. C.
Miles, H. S.
Mittendorf, W. F.
Oihly, John H.
Parker, Walter R.
Payne, S. M.
Posey, Wm. Campbell
Price, Henry R.
Pyle, Walter L.
Reeve, R. A.
Reynolds, W. G.
Ring, G. Oram
Ring, Henry W.
Risley, J. Norman
Risley, Samuel D.
Sautter, A. C.
Schneideman, T. B.
Schwenk, P. N. K.
Shannon, John R.
Shoemaker, J. F.
Shumway, Edward A.
Smith, E. Terry
Snell, Albert C.
Spalding, F. M.
Standish, Myles
Stieren, Edward
Sutphen, T. Y.
Sweet, W. M.
Tarun, Wm.
Theobald, Samuel
Thomson, Edgar S.
Tooke, Fred. T.
Tyson, Henry H.
Vail, Derrick T.
Verhoeff, F. H.
Weeks, John E.
Weidler, Walter Baer
Wheeler, John M.
Wilder, Wm. H.
Williams, Edward R.
Wilmer, W. H.
Wood, J. Scott
Woods, Hiram
Wootton, H. W.
Zentmayer, W.
Ziegler, S. Lewis

William M. Sweet,
Secretary-Treasurer.

TUBERCULOUS IRIDOKERATITIS—SOME FEATURES IN ITS PATHOLOGY.

CAPTAIN FRED. T. TOOKE, C.A.M.C.,
Montreal, Canada.

Iritis, perhaps more than any other form of ocular disease, has manifested a modification in its classification in comparatively recent years. Even including that evanescent issue, conjunctivitis and its bacteriology, no branch of the subject has made more actual advance than the study of iritis. The text-book of less than two decades ago taught that lues was responsible for 75 per cent. of all patients suffering from iritis, and that so-called rheumatism, gonorrhea, tuberculosis, and trauma had to scramble for the balance. The Wassermann reaction, the complement-fixation test, the careful examination of the sources of autogenous infection, teeth, tonsils, accessory nasal sinuses, and bowel, have modified the classification percentage of earlier days to a very appreciable degree. It is questionable if the figures of Hirschberg, who quotes 12 cases in 60,000, and Terson, 2 in 30,000 eye cases, would stand in the light of our present knowledge. The masterly work of de Schweinitz on uveitis, read before a recent International Ophthalmological Congress, would support such a challenge.

With a preconceived endeavor of exhibiting some of the tuberculous manifestations of the disease I have been peculiarly fortunate, during the past few years, in having material supplied me manifesting tuberculous processes in the iris with other associated features of the disease. I would, at the outset, record my thanks to those of my col-

leagues who have, by their contributions, made this paper possible.

The pathologic manifestations of this subsection of iritis are no less varied than the alterations of our point of view clinically and etiologically of iritis itself. The pathologic manifestations have naturally many features in common with other forms; on the other hand, each has its own peculiar or particular distinguishing feature.

CASE 1.—Material supplied by Dr. J. W. Stirling, Royal Victoria Hospital, Montreal.

Mrs. G., aged thirty-five years, married. Active but poorly nourished, and with a marked family history of tuberculosis. A history of trauma of the right eye fifteen years earlier, the condition of inflammation requiring three months to subside. Present attack had been in progress for three weeks before she decided to consult Dr. Stirling. The cornea was steamy, or semi-opaque, the condition being more intense at certain segments, especially in the deeper tissues. Details of the iris could not be determined, and there was marked pericorneal injection. There was a slight hypopyon but no acute pain. Later the hypopyon became more manifest, and the infiltration of the cornea more intense, with the presence of what appeared to be deposits on Descemet's membrane. There was a general rise of evening temperature to 99.2°; tension was low.

A paracentesis of the cornea was done, and a clot of organized exudate was removed from the anterior chamber without complication. Rapid progress followed this procedure, all signs of inflammation subsided, and in two days' time she was discharged from the hospital.

The specimen supplied resembled a particle of pus or slough. It was irregular in shape, of a yellowish-white color, and of a firm or solid consistence, not disintegrating when immersed in fluid. It measured approximately 6 x 2 mm. Sections were made in paraffin.

On examination by low power the section appears as a ribbon-like band, curved as though one side had been in contact with the inner surface of the cornea. It consists for the most part of two types of cells. The structure is of a

Fig. 1.—Case 1. Stroma of new connective-tissue fibers most dense anteriorly. Scattered through stroma are innumerable leukocytes and several tubercles best noted in the posterior portion of the section (low power).

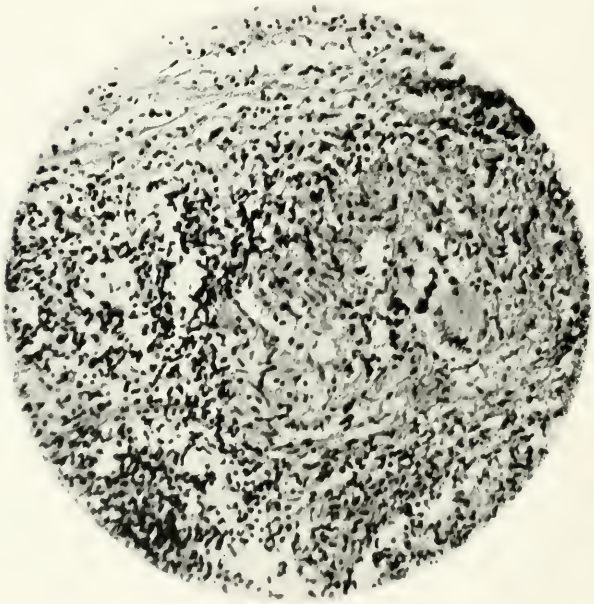

Fig. 2.—Case 1. Section through a tubercle. Infiltration consists mostly of lymphocytes and of a few large mononuclear cells and polymorphonuclears. Epithelioid cells and several giant-cells also evident (high power).

network of new connective-tissue fibers, which are in fairly close apposition at one or two points, but at others the intercellular spaces are fairly well defined. The stroma of connective tissue is most dense anteriorly, bounding that part of the band the curvature of which would appear to conform to that of the adjacent cornea. Scattered through the stroma are quantities of white blood-cells which are distinctly more numerous as one proceeds backward (Fig. 1).

On examination by higher power one frequently observes that these leukocytes are so numerous as completely to occupy the field. They are of the small mononuclear or lymphocyte group, although some polymorphonuclears as well as occasional large mononuclear blood-cells are present. At the end of the section behind the anterior stroma of connective tissue the lymphocytes have formed definite tubercles. They are round, and their periphery is bounded by a dense aggregation of small mononuclear leukocytes. The tubercles consist otherwise of large epithelioid cells, which occupy the center of each tubercle. Numerous giant-cells are present (Figs. 2 and 3).

Although actual necrosis or caseation can hardly be said to have occurred, the cells most centrally placed have begun to show evidences of degeneration, being distinctly shrunken in size and contour. Their cytoplasm stains decidedly less distinctly than the cells more peripherally situated. Several sections were stained for tubercle bacilli, but none could be demonstrated.

An especially careful examination was made by high-power magnification for the detection of endothelial cells, which cover the anterior surface of the iris and line the cornea. That none of these cells could be found, and that the section is quite free from pigment cells, with which the iris is more or less infiltrated during chronic inflammation, precludes the possibility that a superficial nodule has sloughed away from the iris and has formed a second focus of inflammation in the anterior chamber. A more reasonable conclusion would be that of a subacute or chronic inflammation of tuberculous origin, where the exudate, particularly from the ciliary processes, consisted largely of lymphocytes, later to coalesce and form tubercles, which subsequently break down, rather than to treat of them as

pus-cells, as found in an acute hypopyon iritis. The action of light, penetrating a moderately clear cornea, may, in a measure, have arrested the progress of the disease in the anterior chamber, allowing a definite encapsulation of tubercles and lymphocytes more anteriorly to take place.

CASE 2.—Material supplied by Dr. Richard Kerry, Western General Hospital, Montreal.

I. R., a young English girl, aged eighteen years, complained of pain and redness of the left eye with intolerance of light. On examination the cornea at first suggested a condition of a superficial interstitial keratitis. There were areas of grayish streaks which tended to coalesce and to become more dense, with apparent roughening of the corneal epithelium. Under palliative treatment the eye became worse, infiltration of the cornea more intense, pericorneal injection more apparent, and the iris studded with a series of small grayish nodules. There were numerous posterior synechiæ, the pupil dilating but slightly. There was an associated condition of cervical adenitis, presumably tuberculous; evening temperature, 99.2°.

The anterior chamber was opened on two occasions and iodoform powder introduced; iodoform emulsion was also administered hypodermically. The eye was subsequently enucleated by Dr. Kerry, as it was practically sightless, owing to corneal involvement and to the cataractous condition of the lens incumbent on the generalized posterior or ring synechia of the iris.

The globe was fixed and hardened in the usual way and subsequently embedded in celloidin. The section shows very little evidence of acute or subacute inflammation at the corneoscleral margin, and the vessels are not excessive in number or altered histologically. There is no apparent infiltration about the walls of the blood-vessels, and the corneal epithelium shows no manifestation of edema or areas of exfoliation. Bowman's membrane is intact. Throughout the substantia propria there are innumerable areas of infiltration, which are most numerous near the limbus of the cornea. Each individual area is small, and does not appear to occupy more space than that of a single corneal cell, or at most of two cells. At no point do these

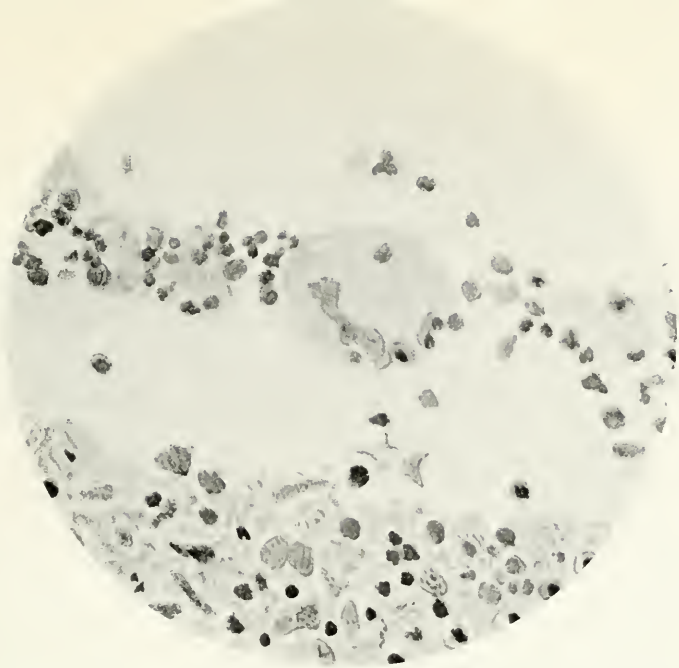

Fig. 3.—Case 1. Practically same as Fig. 2, showing giant-cells, leukocytes, fibrinous exudate, and endothelial cells lying free in the anterior chamber. (Oil immersion $\frac{1}{12}$.)

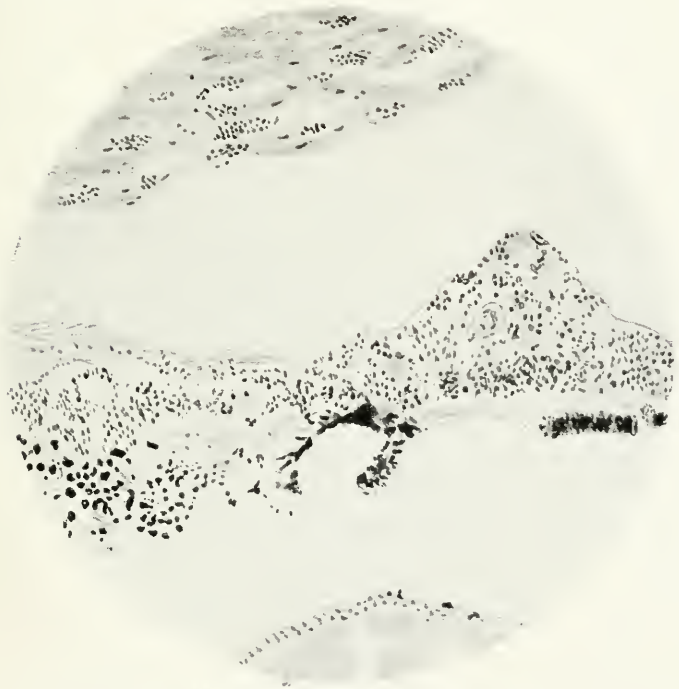

Fig. 4.—Case 2. Marked infiltration of iris stroma with lymphocytes. Tubercle formation with giant-cell near the pupillary margin of the iris. Pigment layer torn away posteriorly with evidence of its earlier attachment to anterior lens-capsule.

assume a rounded or miliary appearance. The infiltration consists for the most part of small mononuclear leukocytes. There is no evidence of giant-cells or of caseation in the cornea.

Descemet's membrane shows no presence of punctate bodies on its endothelial surface. There are areas where dense bands of adhesions have extended from the iris to Descemet's membrane, and where the latter has been ripped away by inflammatory bands extending from it to the anterior surface of the iris. These bands consist of inflammatory connective-tissue cells, of various forms of leukocytes, and in some instances with giant-cell association, typical anterior synechiæ.

The tear or separation of Descemet's membrane I interpret as mechanical, probably the result of opening the anterior chamber for the introduction of the iodoform. The iris is markedly infiltrated with large and small mononuclear leukocytes, as well as by some polymorphonuclears, to such a degree that details of the iris stroma and of its component vascular arrangement cannot be determined. Definite tubercles are scattered over the anterior surface of the iris, mostly over the pupillary margin. These consist of fibrinous material and of lymphocytes, with occasionally epithelioid cells, and others in which an early stage of necrosis may be said to have begun. Giant-cells are present. There are no evidences of tubercle about the filtration angle, although one can be detected posteriorly midway between the pupil and the root of the iris (Fig. 4).

The pigment layer of the iris is distinctly disintegrated, and is thrown off into the posterior chamber. It has been collected at certain foci as definite masses, and when plastered down to the anterior lens capsule forms dense posterior synechiæ, particularly about what was once regarded as the pupillary margin. At a greater distance from the pupillary margin these synechiæ are less dense, and an iris bombé is clearly shown, with occlusion of the filtration angle.

Cataractous changes have occurred in the superficial lamellæ of the lens cortex. A mild degree of infiltration is noticed about the ciliary processes, with numerous erythrocytes lying free in the vitreous chamber. The retina

and choroid show no associated inflammatory changes. Tubercle bacilli could not be demonstrated in the tissues.

CASE 3.—Material supplied by Dr. W. G. M. Byers, Royal Victoria Hospital, Montreal.

M. Y., female, single, aged twenty-seven years, consulted Dr. Byers for pain and inflammation in the left eye. She gave a history of the eye having been struck by a splinter of wood a month previously, but that it gave her no trouble for three weeks' time. It then became red and she could not stand the light. The patient was a very poorly nourished woman, and although a physical examination was carefully made, no evidence of a general tuberculous lesion could be found. There was no history of tuberculosis in her family.

There was a small central opacity of the cornea, with considerable hyperemia of the iris. There were numerous deposits on the inner surface of the cornea, apparently mostly central and to the temporal side. The pupil could be kept fairly well dilated. The cornea tended to assume a steamy or edematous appearance, and there was distinct tenderness over the ciliary body. There was no manifestation of tubercles of the iris, but subsequent examination was finally inhibited by a generalized interstitial keratitis.

Approximately one month after admission to the hospital the patient showed marked signs of improvement, although the cornea did not clear up. The evening temperature was 99°, morning 97.3°. She was finally discharged, as she had an opportunity for convalescence in the country.

She was readmitted to the Royal Victoria Hospital two months later with the eye in an acutely inflamed and painful condition. The cornea was completely opaque and somewhat staphylomatous. Central ulceration had taken place, and a definite hernia of Descemet's membrane was evident, measuring less than 2 mm. across. She said that this had burst on more than one occasion. Details of the interior of the eye could not be obtained. The eye was naturally soft, and vision was reduced to perception of light only. There were no changes in her physical condition, and the temperature remained as at last entry. A smear of the conjunctival secretion showed a few Gram-positive cells, later staphylococcus albus. The eye was enucleated by Dr.

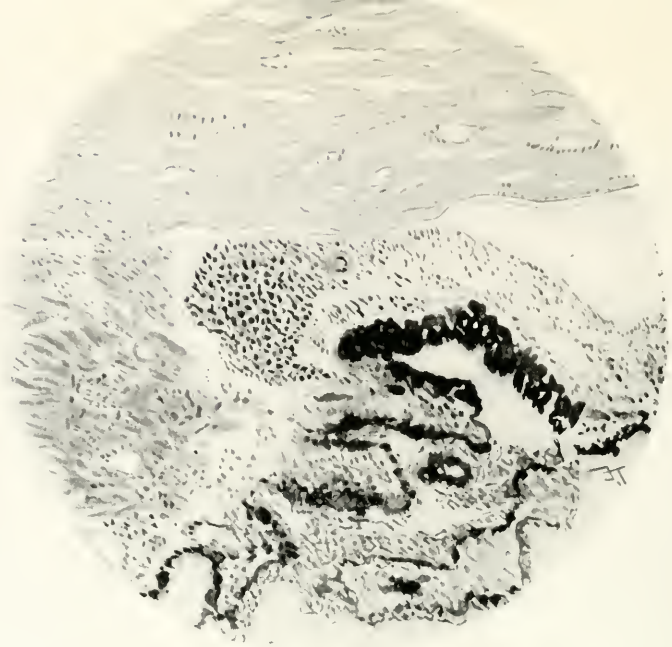

Fig. 5.—Case 3. Marked infiltration of iris stroma with definite tubercle formation at root of the iris, showing giant-cell. Moderate degree of injection of ciliary body with infiltration of leukocytes about the ciliary processes.

Fig. 6.—Case 4. Taken near the filtration angle. Gradual necrosis of Descemet's membrane with groups of leukocytes adherent to its endothelial surface. Above it and between the deeper cells of the substantia propria is a marked infiltration of leukocytes with giant-cell formation.

Byers. A von Pirquet reaction subsequent to enucleation was positive.

On account of the necrotic condition of the cornea, this is the most complicated but by no means the most interesting of the sections. There is a marked condition of superficial as well as of deep injection of the pericorneal vessels. The deep vessels especially show a marked condition of dilatation, with clumps of leukocytes aggregated about the vessel-walls. The superficial layers of the corneal epithelium are multiplied, and the deeper layers are edematous. The epithelium is distinctly irregular, dipping down into the diseased substantia propria as definite bud or nipple-like projections. Bowman's membrane is intact, but wavy and irregular in its course. Doubtless it is broken in other sections. Directly beneath this the cornea is distinctly vascular—naturally more so at the corneoscleral margin.

The substantia propria is markedly necrotic, the intercellular spaces being crowded with lymphocytes and with some pus-cells. There are tiny blood-vessels in the cornea, the more superficial areas showing a condition of perivasculitis. A definite tuberculous nodule is well seen about the center of the substantia propria, fairly close to the filtration angle, and about midway between Bowman's and Descemet's membrane. It stains somewhat pale at the center, but there is no evidence of necrosis or of giant-cell formation. The center of the cornea is distinctly necrotic, being occupied entirely by granulation tissue. The granulation consists of a stroma of amorphous cells in which are included fibroblasts, lymphocytes, and the various elements of a chronically inflamed iris, pigment cells, and a mass of endothelial cells. Descemet's membrane is naturally broken and curled up at the point where the earlier ulcer perforated. Where it is intact, numerous aggregations of lymphocytes line its endothelial surface. Numbers of leukocytes and fibrin lie free in the remains of the anterior chamber.

A definite tuberculous nodule with a central giant-cell is apparent in the filtration angle, while another is even more evident in the ciliary body of the same side, close to the root of the iris (Fig. 5). The iris is injected and shows small tubercles in the process of formation in its stroma nearer the filtration angle. So much of the iris is necrotic and

amorphous, included in the ulcerated process of the cornea, one may reasonably suppose that the presenting portion may have been formerly more extensively diseased. There are, however, no giant-cells in the portion of the iris included in the diseased cornea.

The ciliary body is moderately injected, the lining cells of the ciliary processes showing a manifest exhibition of leukocytes attached to their side, while other cells lie free in the neighboring vitreous, at least one forming a distinct clump—the nucleus of a later tubercle. Some of the retinal vessels are distinctly dilated, with perivascular exudate about their walls and with numerous leukocytes, chiefly lymphocytes and polymorphs within the lumen of the dilated capillaries. A perivascular tubercle is well shown in at least one section. There is only a very moderate degree of infiltration in the stroma of the choroid.

CASE 4.—M. M. C., female, single, aged forty-six years, consulted me complaining of pain and loss of vision in the left eye. The condition developed suddenly seven years previously, when she was out-of-doors, at which time she experienced a sensation as of a grain of sand having blown into her eye. The eye recovered, but broke out with similar attacks on a number of occasions. It became perceptibly worse six months ago, when variations in temperature and bright lights would cause her a great deal of pain and photophobia. She was referred to the Royal Victoria Hospital.

The patient was an unusually large but neurotic woman. Her previous health and family history were good. There was no evidence of focal tuberculosis elsewhere in the body after a very careful physical examination. The eye was moderately injected, with intense intolerance to light. The iris was discolored and would not dilate to any appreciable extent. There appeared to be no deposit peripherally on the endothelial surface of the cornea. The central portion of the cornea, or that considered as the area covering a moderately dilated pupil, was occupied by a dense, regular, deep-seated grayish mass. The condition was diagnosed as a tuberculoma of the cornea, and a diagnostic dose of 2 mg. of T. O. was given. The temperature rose from subnormal

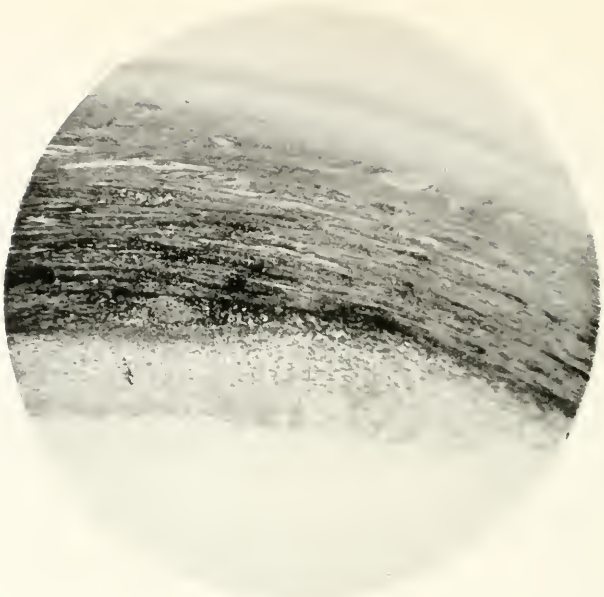

Fig. 7.—Case 4. Intense infiltration of substantia propria, especially the deeper layers. Lowest layers of cells actually necrotic, stain poorly, and innumerable giant-cells are manifest (low power).

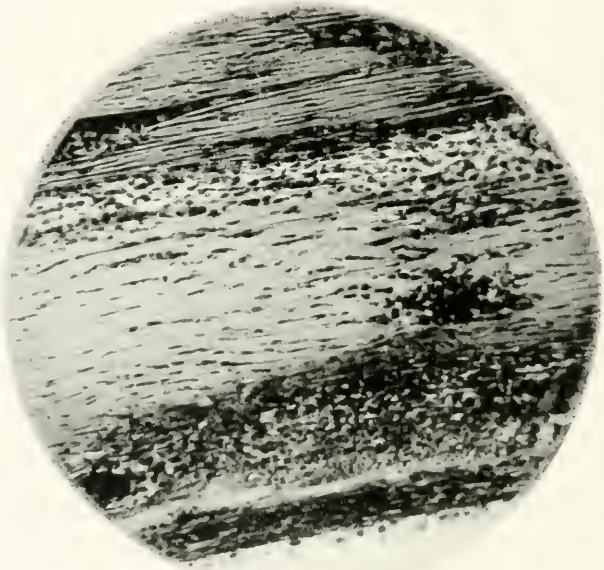

Fig. 8.—Case 4. Shows character of infiltration of cornea. Deeper layers are actually necrotic, with area of infiltration assuming the form of a tubercle above. Necrosis of Descemet's membrane with marked proliferation of endothelial cells (high power).

to 99.2°, and an acute reaction occurred in the eye. Therapeutic doses of tuberculin emulsion were then proceeded with, starting at $\frac{1}{20,000}$ mg., but no improvement ensued. As the eye was practically sightless, and the pain was intensely acute, I enucleated the eye at the patient's request.

There is marked superficial and deep injection about the limbus corneæ, the deeper vessels showing a decided infiltration about the vessel-walls. The more superficial veins are widely dilated and are filled with red cells. There is no edema of the corneal epithelium. Bowman's membrane is intact and, comparatively speaking, the superficial lamellæ of the substantia propria are clear. As one proceeds more deeply there is an intense infiltration of the substantia propria (Fig. 7). This is so evident at the center of the section in the neighborhood of Descemet's membrane that actual necrosis is seen to have occurred (Fig. 8). The infiltration throughout the cornea consists practically entirely of lymphocytes. In the very deep layers these have broken down and stain very faintly, constituting what has to be regarded as practically amorphous cellular tissue. Scattered throughout this area are innumerable large giant-cells.

Beneath this, at the center of the section, Descemet's membrane is also necrotic and cannot be recognized as such. It is clear, but lined with organized lymph or possibly by proliferated endothelial cells. There are numerous leukocytes or clumps of leukocytes attached to its endothelial surface at the more peripheral parts of the section.

The iris is markedly infiltrated, and several small nodules can be seen on its endothelial or anterior surface. One nodule practically occupies the filtration angle at one point, while others may be recognized at the pupillary margin. There is no necrosis or evidence of giant-cells in the iris, but the iris pigment is markedly disintegrated. The iris is intensely injected, and at one point has fallen forward, completely occluding the filtration angle. There is considerable inflammation of the ciliary body, and the more anterior of the choroidal vessels show a decided injection about their walls.

Parsons classifies tuberculous lesions of the iris as follows: (1) Miliary tubercle; (2) confluent or conglomerate tubercle; (3) tuberculous iritis. The last type may be indistinguish-

able clinically from other forms of chronic iritis, but microscopically sections reveal tubercle systems or the presence of bacilli.

The four cases just recorded may be considered to fall into the last-mentioned class, not only on account of the clinical appearance of the various lesions, but also because of the peculiar chronic nature of each individual case when compared with the more acute lesions of classes 1 and 2. Parsons considers class 3 to be the rarest form of the disease in the iris, and refers to cases of Costa and Prunenda, Edmunds and Brailey, Knaggs, and Kalt. There is generally diffuse infiltration and thickening of the iris. Giant-cells may be present, but tubercle systems not necessarily evident. Naturally necrosis with an exhibition of pus in the anterior chamber supports the diagnosis. The demonstration of tubercle bacilli is, naturally enough, conclusive evidence, but the diagnosis is not necessarily excluded on account of the inability of demonstrating the microörganisms. Animal inoculation may be of value. Diagnostic doses of tuberculin, in my own clinical experience, are not always necessarily positive.

Comparing the tuberculous with the syphilitic nodule, the opinion has been that the last-mentioned occupies the root of the iris. Our more exact methods of diagnosis, as well as some of the more recent pathologic contributions, make us somewhat more guarded in indorsing such an assertion. For, if the statement be reversed, we note in case 2 of my series that the lesion appears anteriorly near the pupillary margin, but a second nodule may also be detected directed backward, and not far from the root of the iris. In case 3 a nodule is definitely at the root of the iris, and one also in the ciliary body, with a secondary or, more correctly speaking, associated tubercle in the substantia propria. In case 4 the infiltration is so intense that actual tubercle systems cannot be detected in the iris.

It is interesting to record in this regard that within the past three months I have had two patients with iritis developing very suddenly, and from no apparent cause. In each case the nodule or nodules of the iris were quite evident about the filtration angle below, and an associated keratitis was present to an alarmingly acute degree. A Wassermann reaction of the spinal fluid was negative, but a positive reaction, both local and general, to tuberculin, in one case to 0.5 mg., in the other to 2 mg., added confirmatory evidence to a suspected diagnosis.

In the syphilitic nodule the iris tissue itself is of normal thickness, and the nodules fade away in the surrounding tissue. They may break through the surface of the iris and communicate with the anterior chamber. They consist of mononuclear cells embedded in a fine reticulum. As in gummata, giant-cells may be found, but rarely caseation. There are widely dilated capillaries within the nodules, while tubercles, on the other hand, are avascular.

No little interest is shown in the discrepancy of opinion as to whether the tuberculous focus in the iris is primary or secondary to a preëxisting tuberculous process. Leber, Fuchs, and de Wecker claim the disease invariably to be a secondary manifestation, while Lawford, Parinaud, Bach, and others are as positive of the iris itself being the initial source of infection. In case 1, although no primary source of infection could be actually pointed out, a very suggestive family history of tuberculosis would make one more than suspicious of a latent tuberculous lesion. Case 2 showed systemic manifestations of the disease in inflamed glands of the neck. Case 3, although the most suggestive in appearance of all four, showed no actual lesion after careful clinical examination, but exhibited a positive tuberculin reaction after the eye had been removed. Case 4 was an unusually robust, heavy woman, but gave a very marked

local reaction to 2 mg. of T. O., with a corresponding general reaction.

It is further of interest to note that each of my cases was a female, ages ranging from eighteen to forty-six years. Seven out of eight human cases reported by Stock were also females, the manifestations of the disease appearing still later in life.

One cannot approach an intelligent appreciation of tuberculous iritis without recognizing the painstaking and scientific endeavor manifested in the work of Stock, on the one hand, and of Verhoeff, on the other. The latter has seen fit to disagree with the former in his work on the experimental production of intraocular tuberculosis in animals, Verhoeff preferring to inoculate dead bacilli or the toxins of bacilli directly into the anterior chamber or into some other part of the eye. Stock, on the other hand, preferred a mild dilution of the actual bacilli, which he introduced into the ear veins of rabbits. Although Verhoeff's technic would at first glance suggest a more benign form of infection, as possibly more comparable with that exhibited in the actual process as it exists in man, yet Stock, by introducing the bacilli at a point away from the eye, allowed for a liberal dilution, also for such inhibition which the lymphatics and leukocytes might supply. It is further questionable if, when the microörganism reached the eye, the reaction was more violent in the one series of cases than in the other. We must also not overlook the tremendous care and completeness of Stock's undertaking. I can vouch for its thoroughness, having been with him when part of it was in progress. Forty-eight animals were inoculated, and a careful clinical and pathologic examination, not only local, but also general, was systematically made of each individual case. That the effect of inoculation was not invariably unusually acute is proved by the fact that some rabbits lived up to two years after inoculation. Further-

more, in cases where tuberculous iritis was induced, healing in some instances took place in from two to five weeks.

Stock, from his experimental work on rabbits, has come to the following conclusions: The first symptom manifested is a general thickening of the iris tissue, without any unusual thickening of the blood-vessels. Three or four days later small grayish masses appear in the iris tissue. As I have already given it as my clinical and pathologic experience, Stock supports my contention by affirming that there is no such thing as a site of predilection for the tubercle of the iris. It may appear at the pupillary margin, at the middle of the iris, or at the filtration angle. The nodules on healing leave a white spot. Some animals showed a generalized hyperemia of the iris, with the formation of new vessels and a general appearance of granulation tissue. Infiltration may be so intense that solitary nodules cannot be detected. Later there may appear changes in the surrounding tissue, as parenchymatous keratitis, with manifest pericorneal injection of the deep ocular vessels.

Pathologically the nodules, though possibly having a site of predilection, are more numerous on the posterior than on the anterior surface of the iris. The nodules consist at first of mononuclear leukocytes without any epithelioid cells. Some nodules may reappear over the site of a previously healed tubercle. When left for a long time and allowed to increase in size, one notes in the center of the nodule large mononuclear cells with pale staining nuclei—epithelioid cells. One can only find giant-cells in those cases where the process has been active for months, and tubercle bacilli could be demonstrated only in those cases where central caseation of the individual tubercle had occurred.

In the more malignant form, where a parenchymatous keratitis ensued, the iris was generally studded with tubercles. The center of such tubercles was generally necrotic. The sections, especially those from cases 3 and 4, show a

distinct disturbance of the endothelial cells of the cornea when a keratitis punctata is instituted at the back of the cornea. In the animals inoculated, as well as in the human cases recorded by Stock, Descemet's membrane always remained intact. Case 4 in my series is rather an exception to this rule or acceptance, where a fairly distinct atrophy or actual necrosis of this membrane may be seen to have taken place. Descemet's membrane behind is densely covered with lymph, and above by a distinct infiltration of lymphocytes and numerous giant-cells in part of the cornea suggesting an early necrosis. On the other hand, case 3 demonstrates the absolute obstinacy and resistance of Descemet's membrane, presenting itself forward as a knuckle or hernia or descementocele after an actually complete ulceration of the substantia propria has taken place. The resistance of this form of hyaloid membrane is quite comparable to that already pointed out by me in the case of the membrane of Bruch in cases of tubercle of the choroid. The whole cornea may be diffusely infiltrated, and new vessels may encroach upon the corneal tissue. There is a circumscribed nodule formation in the cornea itself, as well as about its margin.

An infiltration of the cornea is the rule rather than the exception in my experience. The ciliary body is not of necessity always acutely involved in the diseased process. In gross involvement of the iris one may find large or small tubercles of the ciliary body. Stock was unable to find the same pathologic manifestations in animals as one would expect to find from clinical observations in the human.

This article would be far from complete were I not to attempt to acknowledge the important scientific work done by Verhoeff. His contribution on animal inoculation, as well as his reports on human tuberculous eyes pathologically examined, is more or less restricted in comparison with Stock's work. But his general approach of the subject and

his astutely scientific interpretation of the possible transmission of the disease is more than on a par with the work of earlier writers. By introducing dead bacilli into the vitreous, or, for that matter, into the anterior chamber, he has shown that in several weeks, or even in months of time, there may be produced nodules in the corneosclera closely simulating those of sclerokeratitis, as is manifested clinically in man. These have been manifested in a naturally modified form in all my cases, with definite lesions in the iris and ciliary body, and also in the retina, as I have been able to demonstrate in one case. In the case of a marked involvement of the cornea little reaction may be apparent in the ciliary body, a condition interpreted to the fact that live bacilli, even though of an attenuated virulence, will find the cornea and sclera more suitable for their proliferation owing to their avascular nature. The cornea is more involved in experimental work on animals directly inoculated, while the reverse is the case in man. This is as one would suppose or expect—in the one case the inoculation is almost direct through the medium of the anterior chamber, in the latter instance it is indirect through the venous capillaries of the uveal tract.

Verhoeff makes the statement that in his experimental cases the size and number of the lesions were greater at the filtration angle than elsewhere, showing that more bacilli lodged there. One might offer as an explanation the fact of the prevalence of tubercles of the iris occurring close to the filtration angle as borne out by Stock, as well as by myself. Sections from the cases which I am presenting do not necessarily always substantiate this fact, although acknowledging the almost constant association of corneal involvement with tuberculous iritis. In the cases here recorded the iris itself is more involved than any other part of the eye. Furthermore, we notice in this series, as well as in the cases which I have had an opportunity of studying clinically,

that the lesion in the cornea is more often opposite the pupillary area than about the filtration angle and ligamentum pectinatum. One may explain such a condition by the fact of the toxins ultimately reaching that part of the cornea with the most stagnant of lymph supply, as one notices in any form of interstitial keratitis. Further, the aggregation of lymphocytes, with the attendant bacilli, are drawn directly from the posterior chamber through the pupil to the anterior chamber. From albuminous changes in the aqueous and consequent alteration in its specific gravity these lymphocytes, instead of falling down into the filtration angle, fall more directly forward. Such a condition of so-called punctate keratitis, though not perhaps an actual source of infection, has at least an influence in inhibiting nutrition from the aqueous, which we are led to believe supplies Descemet's membrane and the posterior layers of the cornea. These layers, with Descemet's membrane, are in consequence attacked from both sides. This interpretation has been shown to be more than a probability in cases 3 and 4.

There was evidence of an active or acute inflammatory condition in the choroid in one case only—case 4. Case 3 shows what might in time have developed into a retinal tubercle—a widely dilated vein filled with numbers of leukocytes, many, strangely enough, being of the polymorphonuclear type. There is distinct evidence of the migration of blood-cells through the vessel-walls and of the formation of an early though definite perivascular tubercle. Verhoeff, in his work, very properly supposes or suggests the probability of the infection, particularly as applied to the corneosclera, as coming from the lymphocytes escaping from lesions occurring in the choroid, retina, or ciliary processes. These clumps of cells, actually nascent tubercles, find their way through the pupillary area, later to attack the cornea and corneosclera through the ligamentum pectinatum. The infection is consequently direct and not one of metastasis.

In a former contribution which I had the honor of presenting before this Society I attempted to show how, in cases of tubercle of the choroid, the condition extended through the blood-stream. The migration of lymphocytes through the vessel-walls and the subsequent formation of a perivascular tubercle were demonstrated not only in the vessels about the base of the brain, but also in the pial capillaries extending into the vaginal space about the optic nerve. What is true in the acute form of the disease may, I claim, be equally true in the more chronic phase, though from its mere chronicity not so easily demonstrable. One may thus account for tubercles found free in the vitreous, about the ciliary processes, in the choroid, and even in the more anterior portion of the retina in tuberculous iridokeratitis.

My observations in cases 3 and 4, while maintaining that the infection of the cornea is due to toxins and are endogenous, one may still support Verhoeff's maxims. The aggregation of lymphocytes, or, what I have formerly termed them, nascent tubercles, after reaching the aqueous may find their way to the filtration angle and through the ligamentum pectinatum, the toxins, or possibly the actual bacilli, to be taken up by the pericorneal lymph-capillaries. A condition of parenchymatous keratitis may exhibit actual tuberculous elements other than ordinary manifestations of interstitial inflammation. I do not consider that the necrosis of Descemet's membrane in case 4 has resulted from direct apposition of tubercle upon its endothelial lining, any more than I have been able to demonstrate a diseased process in the membrane of Bruch in the acute form of the disease involving the choroid. My opinion is that a deep interstitial keratitis must first be present, with subsequent necrosis and doubtless with actual bacilli present rather than toxins. Case 3 supports this opinion, as well as the large series of cases prepared by Stock, who was unable to

demonstrate a single case of initial necrosis of Descemet's membrane. Compared with his exhaustive series, both comparative and human, my own exception in case 4 would appear as a unique condition.

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DISCUSSION.

DR. F. H. VERHOEFF, Boston, Mass.: I should like to congratulate Dr. Tooke, in the first place, for availing himself of the opportunity to examine these cases microscopically. There have been remarkably few cases of tuberculosis of the eye of this type examined. There must have been a great many eyes removed, and why they have not been examined it is difficult to say.

I think that Dr. Tooke gave a rather wrong impression in regard to the criticism that I made of Stock's work. I did not criticize in any way his technic or the accuracy of his observations, but I criticized the whole plan of his work. In my opinion his work has absolutely no bearing on chronic ocular tuberculosis as we see it in man. He took normal rabbits and injected virulent bacilli into their blood, producing metastatic foci in various parts of the uvea; but the character and situation of these lesions were different from those in the eyes of human beings that we have examined microscopically. The conditions are entirely different. In the man, the individual has had tuberculosis for a long time, and the tissue-cells and bacilli have developed relative tolerance toward each other. According to Stock's work, we should have to assume that the lesions in the man are due to direct blood metastasis, whereas this is not really the case. My

work was based on the supposition, indicated by microscopic examination, that the lesions of chronic ocular tuberculosis in man are due to bacilli which get into the aqueous humor and, from there, are deposited in different parts of the eye, notably in the filtration angle and on the surface of the iris. From the filtration angle metastases are carried into the cornea and subchoroidal space. It is practically impossible to reproduce in animals exactly the conditions found in man. By injecting dead bacilli into the aqueous or vitreous humors of rabbits, however, I obtained lesions having the exact situations of those found in man.

DR. JOHN E. WEEKS, New York City: I should like to call the attention of the gentlemen to the rather strong resemblance of certain cases of so-called disciform keratitis to tuberculous lesions of the cornea. In the study by Dr. Tooke he tells us that on the posterior surface of the cornea there is either a multiplication of the endothelial cells or a collection of lymphocytes, or both, giving the appearance of deep-seated punctate keratitis. In the two or three examinations of corneas with disciform keratitis the pathologic findings have been those of small-cell infiltration with a caseous degeneration or necrosis of portions of the infiltrated corneal lamellæ and a disturbance of the epithelium. Those who have studied this form of keratitis to any extent, particularly those who have been fortunate enough to observe a case at its inception, find that the infiltration takes place in the superficial lamellæ immediately beneath Bowman's membrane. The infiltration extends downward, and after this condition has existed for a time one or two whitish necrotic areas develop in the parenchyma of the cornea, and, in some cases, a collection of cells and fibrin forms on the posterior surface of the cornea. These collections have been studied microscopically and found to consist of deposits such as those that Dr. Tooke has mentioned. Dr. Tyson, in 1915, advanced the opinion that disciform keratitis was tuberculous, and recent clinical studies of some cases have caused me to arrive at the same conclusion. In my opinion the infection is exogenous. The insensitiveness of the cornea that always takes place in these cases is due to the absorption of toxins and their effect on the terminal filaments of the fifth nerve.

SOLID EDEMA OF THE FACE.

(Report of Three Cases.)

WALTER BAER WEIDLER, M.D.,
New York.

Solid edema of the face was a comparatively new disease to me until I found the following reference in Allbutt and Rolleston's System of Medicine, Vol. IX, p. 183, describing it "as a recurrent and later a persistent edematous swelling without pitting, and characterized as 'solid edema' by Sir Jonathan Hutchinson, involving the whole or limited portions of the face, such as the eyelids or one of the lips, and has long been known and described under such names as 'recurrent erysipelas' (J. Hutchinson, 1883), 'erysipelas persitans faciei' (Kaposi), 'erythema persitans faciei' (Kreibush), 'lymphangitis faciei' (S. MacKenzie), and as persistent lymphatic edema. There are recurrent attacks at short, long, or irregular intervals, in which the skin swells, sometimes with redness and signs of inflammation, but more often not. The part attacked becomes edematous in such a way that pitting on pressure is absent. In many cases there are no febrile or other constitutional disturbances—only the local discomfort, such as the photophobia, lacrimation, and eczematous eruption about the eyelids and nose, and interference with breathing and inability to cleanse and free the nares from the crusts and scales (as was observed in cases 1 and 3). The first attack is usually the most severe, the later recurrences milder. The edema does not always clear up entirely between the attacks—especially is this true of the upper lip."

Case 1. Showing marked swelling of left lids, upper lip, and right cheek.

Case 1. Showing the patient one year later.

Case 2. Showing great swelling of lips and nose. Some swelling of both lids.

Case 3. Showing swelling of nose and upper lip. Great redness, scabbing of the skin of nose and upper lip.

The etiology is not definitely settled, but the general impression is that the condition is due to a streptococcic infection, and that it resembles the recurrent attacks seen in the other parts of the body, as the arms or legs, where it may induce, in time, a form of elephantiasis. In none of my cases here reported was the streptococci found, but in all the cases either the staphylococcus pyogenes albus and aureus and in case 2 there was also a diplobacillus found.

There is evidently a direct absorption of the toxins elaborated by the streptococci and staphylococci in the nasal mucosa, which is absorbed by the lymphatics, and this in turn causes the edema of the cellular tissues of the lids, cheeks, and lips.

In all my cases the nose has been found to be the source of the infection, and a minute search should always be made in the nose, eyes, mouth, or sinuses for any possible source of the infection. Exposure to cold may favor the attacks.

Diagnosis.—Recurrent localized swellings or edema of the lips, nose, cheeks, and eyelids may simulate angioneurotic edema, and when the whole face is involved, at first glance might suggest myxedema, leprosy, dropsy, or pernicious lymphedema.

Krieg-edema, reported as occurring among some of the Russian soldiers in the present war, affects, as a rule, the legs, thighs, genitalia, and the eyelids. This is thought to be due to underfeeding, and especially to the absence of the fats in the diet.

CASE 1.—Miss R. S., aged twelve years, Russian Jewess, states that when five years of age the eyelids, nose, and upper lip were swollen, with some photophobia and lacrimation, which lasted for a few weeks. At ten years of age there was a recurrence of this swelling, which was more limited to nose and upper lip. One physician, who saw her at the time, said that it was due to a "boil" in the nose, but the school doctor pronounced it erysipelas. About six months before

she came to the Manhattan Eye and Ear Hospital she was treated at the New York Skin and Cancer Hospital for several months, but without relief. February, 1916, when I first saw her, there was a marked swelling of the upper and lower lids of both eyes, severe photophobia and lacrimation, with blepharitis, some swelling of both cheeks, and the nose swollen to about one-half again its normal size. The nares were filled with crusts and scales, with some discharge, and the nose was tender and painful to touch. The upper lip was enormously enlarged, and the affected parts of the face were firm to the touch. There was no pitting on pressure, and there was little or no redness.

The urine, Wassermann, and von Pirquet were negative. The report from the rhinologist gave no material help in making a diagnosis. She was referred to the laboratory to have a culture made of the nose, and the report came back that a pure growth of the staphylococcus pyogenes aureus was found. A vaccine was made and the injections given.

Four days after the first injection an abscess at the site of the injection followed, and was later opened and drained. These injections were continued over a period of about two months, with a gradual subsidence of all the ocular symptoms, and the swelling of the nose, cheeks, and lips gradually disappeared. There was some douching of the nose by the patient, but no other treatment was given after the vaccine was started. I did not see the patient again until the following October, and the face was apparently normal. There has been no acute recurrence during the past two years.

CASE 2.—Miss J. G., aged nine years, Italian parentage, family history negative, has had measles, scarlet fever, and pneumonia. Had been under treatment for phlyctenular keratitis two years previously, and was cured by the use of tuberculin injections. Returned to the clinic with a recurrence of the keratitis, and associated with this condition was a marked swelling of the left cheek, nose, and upper lip, with some slight swelling of the lids. About the opening of the nares was some redness, scabbing, and scaling of the skin. There was some tenderness of the nose on palpation.

The present trouble began in September, 1917, with bleeding from the nose, which was shortly followed by

swelling of the upper lip and later extended into the left cheek and lids. She was treated by her family physician, who gave a tonic and ointments with no improvements. This condition has lasted about five months, with recurrent attacks, but the swelling of nose and lip never entirely disappeared.

A culture was made from the nose, and the staphylococcus pyogenes aureus and a diplobacillus were found and a vaccine was made. Injections were given on the following dates: April 27, 1918, 4 drops; May 4th, 6 drops; May 8th, 8 drops; May 11th, 10 drops; May 16th, 12 drops. The patient was then referred to my service at the Bellevue Hospital for continued treatment. On May 25th 14 drops were given; June 1st, 16 drops, followed by local reaction; June 15th, 14 drops; June 23d, 15 drops. The patient was given atropin locally for the keratitis, with the injection of tuberculin. The eye symptoms were relieved in about a week, and the swelling of the face is gradually disappearing.

During the treatment cultures were made from time to time after she was referred to Bellevue Hospital, and the staphylococcus pyogenes albus was found, but the first made vaccine was continued.

Her brother, who brought her to the clinic, had had recurrent attacks of phlyctenular keratitis and was given tuberculin with relief of the condition. Owing to trouble with his nose, a culture was made, which showed the Klebs-Loeffler bacillus. He was given antitoxin, and referred to the Willard Parker Hospital for treatment, and was discharged three weeks later, at which time his nasal cultures were negative.

CASE 3.—Mrs. F. P. U., aged forty-eight years, family and personal history negative, except for scarlet fever in childhood, and recurrent attacks of a condition that was diagnosed as erysipelas. First attack at fifteen years of age, involving head and face; hair fell out after a very severe attack.

Second attack at eighteen years of age, illness lasting about two weeks, affecting face and head. During the next few years she would have recurrences of the erysipelatous eruptions on face, especially affecting the eyelids, nose, and

mouth. The tear-ducts and nose began to give trouble, causing difficult breathing and more or less constant epiphora.

Nasal inflammation extended to pharynx and larynx, causing a cough the entire year. The nasal and throat trouble has been a constant source of annoyance during all these years. The nose condition became so bad that a radical operation upon the ethmoid and frontal sinuses was advised, but was refused. Paralysis of the facial nerve on the right side about five years ago, which was cured after six months of treatment.

Patient was referred to Dr. George MacKee for a dermatologic diagnosis, by Dr. Lane, of New Haven, Connecticut, who in turn referred the patient to me for a nasal and ocular examination. *X*-ray examination by Dr. MacKee showed the accessory sinuses to be normal. "The only suspicious area found was at the apex of the superior right second bicuspid. This tooth should be carefully investigated."

I found at this time that the entire nose was somewhat swollen, especially across the bridge and where it joins the cheek. Dilatation of the veins about the alæ and the bridge of the nose. There was some redness, with small circumscribed elevations, yellow at the center, but not truly pustular. Considerable redness and marked swelling of the tip of the nose, local heat, and some tenderness on pressure. A considerable number of these papillary elevations present a scaling and glazing of the skin. There was a deeper redness where the affected area joins the more normal tissue of the face, and this extended down along the cartilaginous septum, and involved part of the upper lip, which also was slightly swollen. The redness extended into both nares, with marked swelling of the central septal division. Intense dryness with scales and crusts in the nose, which have existed off and on for the past twenty-five years. Has had more or less distinct nasal trouble during that time, which has progressively grown worse and no treatment seems to have had any curative value, only partially relieving the symptoms. The present attack has existed for about three or four months. Has pain deep in the nose and supramaxillary bones at times.

Ears have discharged since an attack of scarlet fever, and about three years ago was given a "colon bacilli" vaccine, but reacted most violently to the injections, which were continued over a period of three months.

A culture was made from both nares, and a pure growth of staphylococcus pyogenes aureus was obtained and a vaccine was made and the following injections were given: April 29th, 4 drops; May 2d, 5 drops; May 6th, 6 drops; May 10th, 7 drops; May 15th, 8 drops; May 23d, 10 drops; June 3d, 11 drops; June 10th, 12 drops; June 17th, 13 drops; June 23d, 13 drops. There was local treatment of the nose, consisting in a thorough cleaning with an alkaline spray, and applications of a .40 per cent. solution of ichthyol; later a 50-grain silver solution was applied high up on the middle turbinate, with a 100-grain solution of silver about the openings of the nares and on the surface of the nose. X-ray treatment was used once a week. There has been a complete relief of all nasal obstruction, and the skin surface of the nose has lost the scaliness and redness, and appears to be gradually becoming like the rest of the skin of the face.

DISCUSSION.

DR. S. LEWIS ZIEGLER, Philadelphia: I published in 1911 a case of a similar nature, although the apparent underlying cause and the manifestations were somewhat different. There was cellular infiltration of the right cheek, with hemophilic extravasation following chronic constipation, accompanied by a vascular growth that projected from the conjunctiva of the right eye. The condition so nearly resembled sarcoma of the antrum that a general surgeon advised immediate excision of the upper jaw. As I had previously treated her, she was again referred to me for further study. I recalled that I had seen her in a similar attack some years before, and that her history showed a recurrence of this disturbance every seven years. Prior to this last attack she had suffered severely from hyperidrosis, starchy indigestion, and chronic constipation, the stools occurring at intervals of more than a week. The urinary examination was negative. The blood count showed erythrocytes, 4,820,000; leukocytes, 10,300; and hemoglobin, 80 per cent. She was placed on thyroid extract and suprarenal extract, with a liberal use of

cathartics. There was some improvement, but it was slow. As she had marked nasal obstruction, which was increased by the pressure of the extravasation, it was finally decided to cauterize the inferior turbinate and the septal puff. One month later, simultaneous with the opening of the nostril, the resumption of free breathing caused prompt and complete absorption of the hemic infiltrate and, coincidentally, the clearing up of a facial acne that had been present for more than a year.

I recall that the patient was also disturbed when she ate peaches. As a rule, strawberries cause more tendency toward hemorrhage, but in this case there was a question of peaches also as being the cause. I think that Mr. Eales, of Birmingham, first discussed the question of hemorrhage in young persons, especially in cases of chronic constipation. This element is worthy of study, as I do not think there is evidence, at least it was not demonstrable in this case, that there is a bacterial origin. It is something like Mikulicz's disease, several cases of which I presented before this Society some years ago. They improved by the measures used to increase the oxidation, one by removal of the tonsils, another by nasal cauterization, and others by other means.

HISTOLOGIC FINDINGS IN EYEBALLS LOST THROUGH COMPLICATIONS FOLLOWING SCLEROCORNEAL TREPHINING.

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Montreal, Canada.

The following cases are of interest as showing the pathologic changes in complications that apparently must inevitably occur at times after sclerocorneal trephining; and are instructive in that they bring out certain points in regard to the technic of Elliot's operation:

Case 1.—A woman, aged thirty-one years. Failure of sight in the left eye noted for several months; no other sub-

jective symptoms. Pupil, oval in the horizontal meridian, fixed. Practically complete cupping of the optic disc, with marked pallor. V. = hand movements; a field (practically circular) of about 5 degrees remains at the fixation point. T. = 37 mm. Hg. R. E., fundus and field of vision normal; V. with sph. +0.25 ⊂ cyl. +0.75 axis 90° = $\frac{6}{5}$. T. = 15 mm. Hg.

A sclerocorneal trephining was performed on March 2, 1915, with a complete iridectomy, easily achieved; no complications. The woman returned only on December 12, 1915, pregnancy and the birth of a child having made it impossible for her to come to the city at an earlier date. The eye had become suddenly painful shortly after leaving the hospital in March.

Examination.—Eye unbearably painful and markedly congested; bleb collapsed, the broken conjunctival covering being mixed with prolapsed uveal tissue; no a. c. In the area of the coloboma upward, occupying about a third of the space, is a small, tongue-shaped patch of corneal infiltration with its base resting on the limbus. Enucleation.

Microscopically one sees (Fig. 1) an open, apparently filtering wound. There is marked thickening, edematous imbibition, and round-cell infiltration of the episcleral tissue. Toward the scleral side the ruptured and thickened conjunctiva overhangs a very loose fibrillar recticulum. On the corneal side large masses of granulation tissue are seen extending inward, and especially downward, from the thickened and bifurcated epithelial layer.

Behind, masses of pigmented cells are streaming forward into the wound around the promontory of the protruding sclera. On the corneal side an enormous number of proliferated endothelial cells, with granulation tissue, are seen extending along Descemet's membrane in the immediate vicinity of the wound, and forward along the cut end of the cornea to plug the aperture.

The posterior opening of the wound is filled by the dislocated lens, save for a narrow channel on either side between the surface of this structure and the sclera and cornea, respectively. The lens-capsule is ruptured, and large numbers of reparative cells (epithelial as well as endothelial) are seen filling the gap and infiltrating cleavages between the lens-fibers.

Elsewhere one sees a well-marked plastic inflammation of the whole of the uveal tract, with the usual changes. The cornea is markedly crinkled on its posterior surface toward its periphery.

The changes in this case are, in an exaggerated way, those that are characteristic of healing corneal wounds, with a marked response to injury or infection on the part of the uveal tract. In contrast to the second case, the dislocation of the lens and the tearing of its capsule in this specimen are attributable to the sudden reduction of intraocular tension following collapse of the bleb.

Case 2.—A remarkably alert and well-preserved lady of eighty-three years. Gradual failure of vision for a few years, attributed to dense nuclear opacities of the lenses. These made a clear view of the fundi impossible. V. R. E. = fingers at 3 feet; small field of vision only about the fixation point. T. = 40 mm. Hg. V. L. E. = fingers at 20 feet; field of vision complete. T. = 23 mm. Hg.

Sclerocorneal trephining November 14, 1915; no complications, but slight difficulty in performing a partial iridectomy. The patient was perfectly comfortable until the morning of the eighth day, when three short, separate periods, in which flashes of light were seen, ushered in complete loss of sight. Following this the eye became more and more irritable and painful, with greatly heightened tension, necessitating enucleation January 22, 1916.

One sees in this case an ectatic scar, covering, dome like, the site of the trephine opening. In all sections except the one here depicted it is of almost uniform thickness, and composed of newly formed fibrous connective tissue (still largely cellular), derived in part from the sclera, the cornea, and the uveal tract, respectively. It is lined for the most part by the atrophied ciliary body; and its concavity is almost entirely occupied by a vitreous prolapse.

In the section (Fig. 2) a narrow, patent channel, immediately behind the cut surface of the cornea, leads from the posterior chamber to a dilated space directly beneath the conjunctiva. The epithelial covering of the latter is greatly

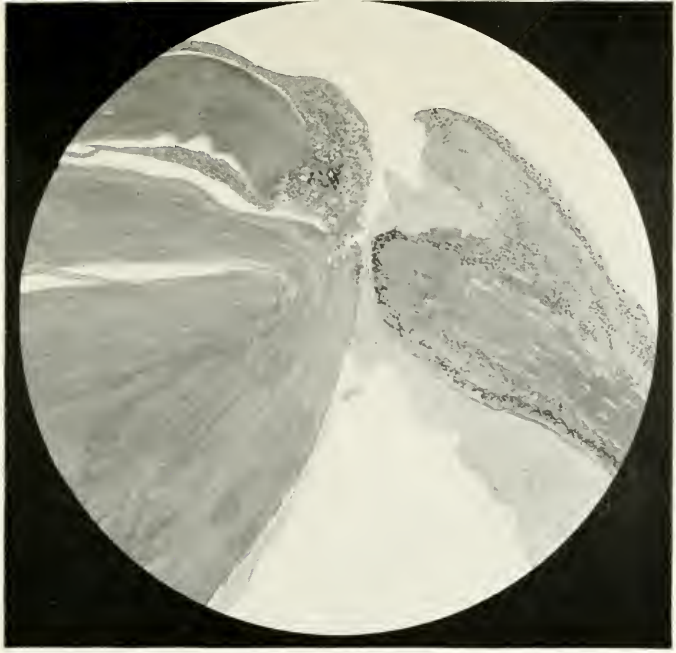

Fig. 1.—Case 1. Section through trephine aperture showing changes which are, in an exaggerated way, those that are characteristic of healing corneal wounds, with a marked response to injury or infection on the part of the uveal tract. The dislocated lens lies against the posterior opening of the wound, and its capsule is ruptured.

Fig. 2.—Case 2. An ectatic scar covers, dome-like, the site of the trephine opening. It is lined for the most part by atrophied ciliary body, and its concavity is occupied by vitreous prolapse. The zonular fibers are ruptured, and the lens is displaced.

attenuated, but the rupture seen occurred during the enucleation of the globe. In this specimen iris tissue is visible along the posterior surface of the cornea; but other slides show that a portion of the iris was actually removed, notwithstanding that elsewhere it is seen greatly atrophied and intimately adherent to the posterior surface of the cornea for a considerable part of its outer continuity.

The lens is displaced downward; the zonular fibers are ruptured; and the vitreous prolapse here also blocks the filtering channel.

The point of interest in this case is the vitreous prolapse, and a brief study of the specimen shows clearly what must have been the course of events leading to its occurrence. The trephining in the first instance was too peripheral, though the operation was performed after considerable experience with the procedure, and it seemed quite satisfactory at the time. During the first few days the uveal tract was sufficiently strong to support the intraocular structures; but it gradually stretched under pressure, and finally gave way on the eighth day, with consequent rupture of the zonular fibers and hyaloid membrane, dislocation of the lens, and renewed tension following vitreous prolapse. At the time of the occurrence of the flashes of light, the feeling was that they were probably attributable to retinal hemorrhages; but there are no extravasations of blood to be seen in any part of the specimen.

The lesson here is obvious. Especially in these advanced cases, in which accidents of this sort are prone to occur, Colonel Elliot's advice in regard to placing the trephine opening in corneal tissue should be followed to the fullest extent. Only in this way can one guard, as far as possible, against the occurrence of the changes described; while, on the other hand, the procedure offers the best chance of securing unadherent iris for excision.

DISCUSSION.

Dr. F. H. Verhoeff, Boston, Mass.: I have examined quite a number of eyes that have been trephined unsuccess-

fully, and in the majority of them I found that the trouble had been an injury to the lens. In a few, which were cases of hemorrhagic glaucoma, in which you do not expect a successful result anyway, the trephine hole had been closed by proliferation of tissue; but in the others the result was due to injury of the lens, either by the instrument or capsule rupture. There was just a small injury to the periphery of the lens, unsuspected by the operator and not discovered until the eye was removed. The whole trouble in these cases has resulted from the irritation caused by the cortical matter as it came from the lens; and it makes a characteristic histologic picture. It produces not an intense reaction, but a marked cellular exudation into the anterior and posterior chambers, as well as in the iris substance, which tends to undergo necrosis. In all cases of this kind, I think, the anterior chamber has become obliterated.

Dr. Walter L. Pyle, Philadelphia: I had recently a case of an unfortunate result from sclerocorneal trephining by a competent ophthalmic colleague. There was an adherent leukoma after prolapse of the iris. I did a posterior sclerotomy, excised the cicatrix, released the iris, and extracted the cataractous lens. I have never performed the Elliot operation in such cases as Dr. Byers speaks of. I see no particular advantage over a broad iridectomy. In these almost hopeless cases I prefer a liberal posterior sclerotomy, releasing a drop of vitreous, and repeating later when necessary.

Dr. Edward Jackson, Denver, Colo.: I have seen two unsuccessful trephinings that seemed to me to be due not to any error in the placing of the opening, but to the character of the cases. They were patients with chronic inflammatory glaucoma that had gone on to practical blindness before operation was done. Although there was no question about the high tension and the glaucomatous nature of the cases at the time of operation, they were probably essentially uveal inflammations in the start, and pursued a course that did not seem to be materially affected by the Elliot operation. They finally quieted down, with high tension, but without causing pain; and neither was enucleated. The fact that impressed me was that the operation, while it gave some relief from pain, did not seem greatly to alter the course of the disease.

CATARACT IN RETINITIS PIGMENTOSA; ITS PATHOLOGY AND TREATMENT.

ARNOLD KNAPP, M.D.,
New York.

It is a well-known clinical fact that a cataract frequently develops in the course of retinitis pigmentosa. Thus Leber* states that this cataract is so constant in the older cases that it often suggests a diagnosis of the condition. The cataract develops in the form of a star-shaped opacity at the posterior pole, limited to the posterior cortical layers. It remains limited to these layers for a long time and rarely develops into a total cataract. In the beginning there is a punctate opacity at the region of the posterior pole, to which one or more radiating opacities are joined, suggesting a star shape. These radiating opacities render the examination of the eye-ground difficult. This cataract is rarely accompanied by other forms of cataract formation; an associated opacity in the anterior layers has been described, and I have observed a well-defined rounded opacity just anterior to the nucleus in two cases of posterior polar cataract in retinitis pigmentosa.

This posterior polar cataract, though it advances but very slowly, seems to be greatly responsible for the reduced central vision of these patients. Doyne† was struck by the remarkable effect produced by the removal of the lens in retinitis pigmentosa, even when the posterior capsule is alone affected and not to a very great degree. Though it is generally believed that the prospect of sight after the removal of the lens is poor on account of the nature of the

* Graefe-Saemisch Handbuch, vol. vii, pt. II. † Ophthalmoscope, 1910.

disease, Doyne states that this is directly opposed to the facts. The improvement that occurs after the removal of the lens, even when so little affected that the normal observer can see every detail of the fundus without difficulty, this author regards as nothing short of marvelous. Doyne has performed the operation several times and, with one exception,—in which central vision was grossly affected,—he was struck by the improvement, which was out of all proportion to his expectations. His experience in this class of cases is so positive that he urges most strongly the removal of the lens in the presence of a posterior capsule that makes a difference in the examination of the eye.

After my attention was drawn by Dr. E. J. Curran, now of Kansas City, to Doyne's views on the advisability of extracting immature cataracts in retinitis pigmentosa, I have been on the lookout for these cases, and I now wish to report on four which have been operated upon by me.

CASE 1.—C. C., aged sixty-five years, was seen June 8, 1914, because the left eye had been failing for four years. V. R. E. = 20/200; field was concentrically contracted to from 2° to 5° from the center. There was the usual posterior polar cataract in the left eye, together with a small opacity apparently on the anterior surface of the nucleus of that lens. The eye-ground presented the usual changes. Wassermann negative. The cataract was removed in the capsule without incident. After operation the vision with correcting glass was 20/70. There were some opacities of the vitreous and an opacity on the hyaloid from a hemorrhage occurring at operation. Subsequent vision was probably better as soon as the opacity was absorbed, but it has been impossible to reëxamine the patient.

CASE 2.—M. S., aged twenty-nine years, has never had good sight, especially for the last four years. V. R. E. = 20/200; L. E. = 20/100. Field contracted to 5° about center, typical radiating posterior cortical opacity in the lens. The eye-ground presented the usual picture. Extraction with capsulotomy was performed, without accident, on the

right eye; after a few days a severe iridocyclitis developed which resisted the usual methods of treatment and did not subside until three abscessed teeth in the upper jaw on the same side were extracted. The resulting thickened secondary cataract was operated upon about eight months later; vision at present with glass is 20/50.

CASE 3.—I. S., aged thirty-two years, always suffered from night-blindness; the sight in the daytime had been failing for four years. Vision is 3/200 in each eye, with the usual posterior cortical cataract and the fundus changes of retinitis pigmentosa. Combined extraction with capsulotomy, no reaction, followed by needling with a resulting vision of 15/200. A thin after-cataract has reformed, which, after another needling, should give still better vision.

CASE 4.—S. P., aged fifty-two years, has been troubled with night-blindness for many years. Gradual diminution of sight in the daytime. V. = 3/200. Posterior polar cataract with advanced changes in the eye-grounds. Field very narrow. The right cataract was extracted in the capsule without event. The result of the operation was excellent, with perfectly clear media. With a correcting glass vision was only 6/200. There was no optical impediment nor particular macular disturbance to explain this poor sight, except that the field was very small and the patient's mentality unusually dull.

To recapitulate, in these four cases,—two of which were operated on by removing the lens in the capsule, and two with capsulotomy,—the visual results were: 1, 20/200, improved to 20/70; 2, 20/200, improved to 20/50; 3, 3/200, improved to 15/200; 4, 3/200, improved to 6/200.

The method of operating required in these cases is not different from that of the usual cataract. The operation is not any more difficult. Doyne observed in every case that he had considerable difficulty in getting the lens away after opening the capsule, as if it seemed to be unusually adherent. I was not able to confirm this peculiarity in the delivery of the lens. After-healing is uneventful. There are no com-

plications as far as the original eye condition is concerned. Doyne has observed that after extracting the lens the capsule has a tendency to form again; a film recurs in the pupil exactly like a wrinkled capsule that requires needling. The visual results are distinctly encouraging, and I entirely agree with Doyne on the correctness of removing the lens under these conditions.

Pathology.—One of the cataracts extracted in the capsule was prepared for microscopic examination with the following findings: The capsular epithelium at the equator of the lens is wavy in outline; the cells are enlarged and have lost their outline; their protoplasm is granular. Underneath this layer there are irregular vacuoles and the adjoining nucleated lens-fibers present vacuoles and are generally irregularly distributed. At the epithelial boundary the new formation of lens-fibers has taken place irregularly and to an increased degree. A thin band of elongated epithelial cells continues along the posterior capsule. Directly internal to this are nucleated cells which are vacuolated, distended, and have proliferated irregularly, often forming nucleated lens-fibers. Just at the center and occasionally above and below the center there are irregular cavities filled with poorly staining lens detritus, albuminous material, globular bodies, and the edges of the bordering lens-fibers are corroded; these cavities represent the lenticular opacities seen with the ophthalmoscope.

The changes, therefore, found in this form of cataract are changes in the equatorial capsular epithelium, prolongation of transformed capsular epithelium along the posterior surface, and irregular cavities of lens detritus in the posterior cortical layers.

The literature contains but few accounts of microscopic examinations of cataracts occurring in retinitis pigmentosa. The best description is that given by Wagenmann,* in which

* Wagenmann: "Pathologische Anatomie d. Retinitis Pigmentosa," Arch. f. Ophth., vol. xxxvii, 1891.

similar changes to those found in the above-described specimen are given.

DISCUSSION.

DR. WALTER L. PYLE, Philadelphia: I should like to ask Dr. Knapp the effect of the operation on the visual field.

DR. KNAPP: The fields showed a concentric contraction to from 2 degrees to 5 degrees about the fixation point. There was no improvement in the field after operation.

DR. PYLE: I had no improvement in the field either.

DR. HERBERT HARLAN, Baltimore, Md.: I should like to report a case that I operated on for cataract about twenty-five years ago. The patient, who was in the Treasury Department at Washington during the Civil War, had long known that he had retinitis pigmentosa. He had an opportunity to go to London, and consulted several oculists there, and had seen the elder Knapp and others; and they all told him that he would eventually go blind. He had been training himself for blindness for years so carefully that he was able to keep his position in the Pension Bureau through the various administrations. He knew the details of every case that had ever come before him, so that it was not necessary for him to look them up. I saw no reason why the central cataract should not be removed, because it was shutting out the only vision he had. There was no difficulty about the operation. He was a good patient, and he told me that he would keep his eyes closed, so that I did not need to reapply the bandage. When glasses were adjusted, he had nearly 20/20 vision. A month afterward he came for an operation on the other eye. The operation was satisfactory, and the man was living last year, still retaining his central vision, and may be still. He could only sign his name in large letters before operation. It is interesting that vision remained so long afterward, and that the retinitis did not progress to cover the central spot.

DR. W. H. WILMER, Washington, D. C.: My experience in operating upon this type of cases agrees with that of Dr. Knapp. My results have usually been successful. After extraction a very strong lens can be prescribed, using either the ordinary frame or the one devised by Dr. Koller for pa-

tients with defective vision. By holding things very close to the face, the patient is able to continue his work. I have had under observation a number of these patients who are still following their vocation—otherwise they would have been put on the shelf some time ago.

I wish to say that I have recently seen Dr. Harlan's patient, who is still living and holding his clerical position. He has consulted me a number of times in regard to his glasses. While there is still a very thin capsule covering the pupillary area, the result is an ideal one. The man has a sort of epileptiform attacks, high blood-pressure, and other physical disabilities. But he has a bright mind and a fine spirit. His son leads him to his office, where he works all day. The spheric portion of his corrective lenses for reading has been gradually increased until it is now a sph.+20 D. By getting his nose very close to the paper he continues his clerical work. At night, when at home, he uses the Koller frame. In spite of his great physical disabilities, he has a central vision of 20/100+. The operation has been very successful when one considers the many complications.

MICROSCOPIC FINDINGS IN A CASE OF CORALLIFORM CATARACT, WITH REMARKS ON CONGENITAL CATARACTS IN GENERAL.

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(From the Massachusetts Charitable Eye and Ear Infirmary.)

Coralliform cataract was first described as such by Marcus Gunn[1] in a brief communication to the Ophthalmological Society of the United Kingdom in 1895. His description of the clinical appearance of the cataract was as follows: "On focal illumination, rounded and oblong gray and white opacities are seen grouped toward the center of the lens, so as to resemble a piece of coral growing forward and outward

from this point. The oblong opacities represent somewhat conical tubes, and their anterior extremities consequently appear as opaque circles or ovals. There are numerous very fine, iridescent crystals throughout the clear part of the lens."

Since Gunn's report, cases of coralliform cataract have been described by Nettleship,[2] Fisher,[3] Langenhan,[4] Kipp,[5] Harman,[6] and Stieren.[7] Langenhan was apparently unacquainted with Gunn's case, but from his description it seems certain that his case was similar. Judging by the histories of the cases the condition is certainly present early in life, and is presumably congenital, although thus far it has not actually been observed at birth. It seems to remain almost if not quite stationary. Nettleship has shown very definitely that coralliform cataract is hereditary. He relates the case of the Betts family, to which Gunn's patient also belonged, in which 30 persons in four generations were known to have been affected. Two children of this family had lamellar cataract, whereas their fathers had the coralliform variety. Nettleship remarks that there was nothing special in the health of the Betts family. The brother of Fisher's patient had congenital cataract, and Harman reported four cases of "axial" or coralliform cataract as occurring in three generations of one family.

So far as I can ascertain, the case I have to report is the only one in which a coralliform cataract has been examined microscopically.

Arthur B., aged thirty-nine years, was admitted to the Massachusetts Charitable Eye and Ear Infirmary August 9, 1912. The patient states that he first became aware at the age of ten that his sight was imperfect and that it has grown no worse since then. The visual acuity of the right eye is $\frac{5}{200}$, that of the left, $\frac{20}{200}$, and in neither eye is improved by glasses. On examination by oblique daylight illumination each lens shows the same picture. Ramifying out from

the posterior cortex is an irregular, brilliantly white structure, closely resembling a miniature coral. Glistening particles, supposedly cholesterin crystals, are also seen here and there in the lens. The lens substance is otherwise clear and permits a fundus reflex to be seen around the opacities.

On August 13, 1912, I removed the cataract from the right eye. The usual cataract incision was made with a Graefe knife, an iridectomy done, a large piece of the anterior capsule removed with toothed capsule forceps, and the lens expressed without difficulty. Almost no cortical matter remained behind, and healing was uneventful. On August 27th I operated on the left eye in the same manner and the patient was discharged from the hospital September 7th. On October 4, 1912, his visual acuity in each eye with sph. $+12\bigcirc$ cyl. $+1.50$ ax. $180° = \frac{20}{100}$.

PATHOLOGIC EXAMINATION.

Both lenses were fixed in 10 per cent. formalin. The left was retained as a macroscopic specimen, the other was embedded in celloidin, and anteroposterior sections made through its center. Each lens measured 7 by 3 mm. in size, only a small amount of the cortex evidently having been lost at operation.

Microscopic examination of the right lens reveals a remarkable picture. The lens in general is normal, but embedded in it are numerous crystals of various sizes, some of them massed together, others occurring isolated. At the posterior and anterior poles the largest masses, as well as the largest crystals, occur, while toward the periphery the crystals are always small in size and become more and more isolated. The crystals are confined to an area 5.5 mm. in lateral diameter, and, with rare exceptions, only in the central portion of the lens are they found at the anteroposterior limits of the specimen. They show little tendency to follow the direction of the lens-fibers, often lying at right angles to the latter. Immediately around each of the larger masses of crystals the lens substance has undergone marked cataractous changes, showing vacuolization and fragmentation. Otherwise the lens substance is normal, except that here and there it shows peculiar round or oval spots, 0.01 mm. to 0.04 mm. in diameter. The central portion of each of these spots stains in hematoxylin somewhat more strongly than

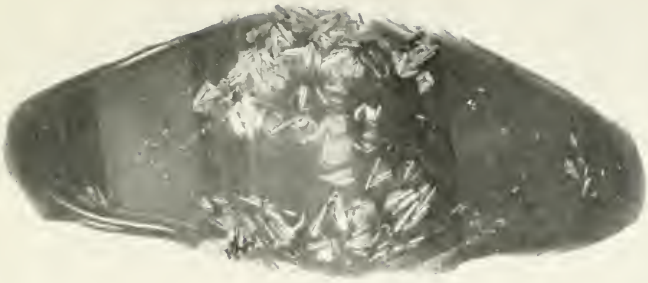

Fig. 1.—Coralliform cataract. Showing arrangement of protein crystals in lens. Hematoxylin and eosin. (Photo × 11.)

Fig. 2.—Protein crystals in lens. Note that degenerative changes in the lens substance occur only in relation with large masses of crystals. Hematoxylin and eosin. (Photo × 50.)

the lens substance, while its periphery stains faintly in eosin, so that the appearance of a ring is produced. These spots occur only near the periphery of the lens and never in close approximation to the crystals or to the cataractous changes around the latter.

When favorably situated in the sections the crystals are seen to consist of rectangular or rhombic plates. The largest seen measures 0.44 mm. in length. The thinnest of the largest crystals as seen in the sections is 0.01 mm. thick, so that it is probable that this is a close approximation to their actual thickness. The smallest isolated crystals are 0.08 mm. long and 0.004 mm. thick. Generally the surfaces of the crystals are parallel, but occasionally one edge is twice as thick as the other. Very rarely a crystal in the shape of a hexagon is seen. This may be the result of an oblique section of a rhombic plate. The crystals injured the edge of the microtome knife only slightly, so that it was possible to obtain sections 5 to 8 μ thick. Numerous cross-lines, however, can be seen on the crystals, produced by the passage of the knife. Scrapings made from the cut surface of the lens also showed the crystals to consist of rhombic plates.

In sections stained in hematoxylin and eosin by the usual method the crystals remain colorless. If, however, the sections are placed in a 0.2 per cent. solution of water-soluble eosin in 80 per cent. alcohol for twenty-four hours, they take a strong eosin stain. The crystals stain most beautifully and intensely, however, in carbolfuchsin. By staining sections twenty-four hours in carbolfuchsin and then differentiating in alcohol and acid alcohol, and finally treating them with a solution of permanganate of potassium, followed by oxalic acid, the crystals are stained intensely red on an almost colorless background. The crystals are also stained by Lugol's solution of iodin. They fail to stain by Gram's method. They also fail to stain in phosphotungstic acid hematoxylin. When placed in a 1 per cent. solution of osmic acid for forty-eight hours, the lens-substance and the crystals are stained the same color—a light brown.

The crystals are found to be insoluble in the cold, in concentrated hydrochloric acid, sulphuric acid, acetic acid, aqua regia, ammonium hydrate, sodium hydrate, acetone,

chloroform, alcohol, ether, xylol, or oil of origanum. The crystals are not melted or affected in any way when sections are boiled in water or absolute alcohol. They are destroyed when sections are placed in a saturated aqueous solution of potassium permanganate for two hours, and then in a saturated solution of oxalic acid for a few minutes. When sections are placed in concentrated sulphuric acid on a slide, and sufficient heat applied to dissolve the lens substance, the crystals are also dissolved. When a section is dried on a slide and then heated over a flame, the crystals are not destroyed until the lens substance is charred and partially burned. When sections are placed in an aqueous alkaline solution of trypsin in an incubator for more than a week, the crystals fail to dissolve. When placed in a solution of pepsin in 4 per cent. hydrochloric acid, they are dissolved completely in forty-eight hours, while the lens protoplasm is only partially digested. When placed in Millon's reagent for ten minutes to twenty-four hours, the crystals are stained an intense rose color, while the protoplasm of the lens substance itself is stained much less strongly. In other words, the crystals give a strong reaction for protein by this test.

Examination of the left lens, which was retained as a macroscopic specimen, also reveals the presence of crystals. This lens was carried through alcohol and xylol in an unsuccessful attempt to make it more transparent. Finally, the xylol was removed and the specimen placed in glycerin. When the lens is viewed under the low power of the microscope, the opacities can be seen to contain numerous transparent rhombic plates.

It is evident that the lenticular opacities in this case were due almost wholly to the presence of crystals. Compared to the crystals, degenerative changes in the lens substance were inconspicuous, occurring almost exclusively around the large masses of crystals, and being absent about the isolated single crystals. From this it seems certain that the crystals were primarily deposited in the clear lens substance and that the degenerative changes probably resulted from their presence. The round spots described as occurring in small number at

the periphery of the lens differ in appearance from any lens changes I have previously seen. They have no resemblance to the vacuoles of congenital cataracts, and never occur in close approximation to the crystals. They somewhat resemble, however, the early degenerative changes of senile cataracts, and are, therefore, probably of the same origin as the latter.

Owing to the arrangement of the opacities in his case, Nettleship assumed that coralliform cataract was identical in nature with the variety of congenital cataract known as spindle, axial, or fusiform cataract, and this view has been accepted by subsequent writers. In my case, similarity to spindle cataract was not noted clinically, but examination of the hardened specimens and the sections shows some indication of a spindle arrangement. Thus the crystals are most abundant in the axis of the lens, and especially at its poles, while peripherally they follow more or less closely the outline of an expanded spindle. Nevertheless, I am convinced that coralliform is entirely distinct from spindle cataract. In the first place, its coral-like appearance is so striking that the appropriateness of the term coralliform is immediately obvious. In the second place the two microscopic examinations that have been made of spindle cataracts in rabbits by Bach and Hess have shown changes that bear no resemblance to those found in my case. Both Bach and Hess found the opacities in spindle cataract to consist of degenerated lens-fibers, and no mention was made of the presence of crystals. From his examination, Hess concluded that spindle cataract was due to delayed closure of the lens sac in the embryo, whereas in my case there was no indication of any malformation of the lens.

The exact nature of the crystals is not at once obvious. Their insolubility in ether, etc., excludes the possibility of their being cholesterin crystals, although in shape they are similar to the latter. Their failure to stain in phospho-

tungstic acid hematoxylin excludes hemoglobin or hematoidin. Their general insolubility and the fact that they stain in carbolfuchsin by the tubercle bacilli method exclude all inorganic crystals. It occurred to me, therefore, that they might consist of the salt of some fatty acid, as calcium stearate. By adding calcium chlorid to an alcoholic solution of stearic acid, rectangular crystals were obtained which had the same insolubility as those of the lens crystals. I was unable to stain them, however, and the possibility of the lens crystals being fatty acid in nature was, of course, completely excluded when I found that they gave the Millon reaction and could be digested by pepsin. That they could not be digested by trypsin is explained by their having been fixed in formalin.

Dr. E. W. Clap suggested to me that the crystals might be protein in nature and similar to protein crystals occurring in plants. It was largely due to this suggestion that the Millon test for protein was applied and found positive. Previously I had presented the problem to several physiologic chemists who were unable to throw any light on the matter. Professor Osterhout suggested to me that the crystals were simply crystallized lens protein, and this suggestion seems to me certainly correct, since it explains their insolubility (after fixation in formalin), their staining reactions,* their positive response to the Millon test, and their digestibility in pepsin. So far as I know, this is the first instance in which protein crystals have been demonstrated in animal tissue of any kind.

The question arises as to the condition under which the crystals become deposited in coralliform cataracts. As already mentioned, Nettleship found two individuals with lamellar cataracts whose fathers had coralliform cataracts. This indicates a close relation between the two forms of cataract. Now it is well known that lamellar cataract occurs

* I find that the protein crystals of a Brazil nut also stain differentially in carbolfuchsin by the method for tubercle bacilli.

most often in children who show signs of rickets, so that it is fair to assume that this form of cataract is dependent upon a faulty calcium metabolism.* It seems more than probable, therefore, that coralliform cataract and lamellar cataract are both dependent directly or indirectly upon a faulty calcium metabolism. It is interesting to note, however, that in only one case of coralliform cataract reported, that of Langenhan, were there signs of rickets. This would tend to indicate some difference in the metabolic disturbances in the two conditions. Microscopic examinations of lamellar cataracts show that the opacities are due to the presence of fine vacuoles. It is evident that neither lamellar nor coralliform cataract is dependent upon degenerative changes, as has been assumed, since in early cases such changes are absent about the vacuoles and crystals. Just what the vacuoles of lamellar cataract contain in the living lens has not hitherto been definitely determined, so far as I can find, but the appearances that they present in microscopic sections indicate with considerable certainty that at some period they contained a fatty substance that has dissolved out. In this connection the following quotation from Stricker's monograph[8] seems to be of considerable significance:

"That there is a chemical difference between the inner and the outer portions of the lens seems to be attested by a fact which, though known for a long time and frequently discussed, has been variously accounted for; namely, that in the lens of all vertebrates and young animals which during life have been perfectly transparent, immediately after death, as soon as the animal becomes cold, the inner portion of the lens becomes cloudy. Michel drew attention to the fact and showed that the cloudiness in the center of the lens of cats, pigs, and calves would disappear if they were warmed

* Lamellar cataract is also sometimes associated with tetany, in which there is believed to be faulty calcium metabolism dependent upon parathyroid insufficiency. Tetany occasionally occurs in rachitic infants, but it is not certain that both conditions are dependent upon the same constitutional factors.

up to 15° to 20° C. This could be repeated as often as desired, and each time the lens would clear up again. This cloudiness is produced by the presence of innumerable roundish, highly refracting droplets in the central portions of the lens, and is not in any way connected with the decomposition of the contents of the lens, such as is produced by freezing. In these experiments there can be no question as to the separation of the water from the albumin. These droplets become less numerous as we go from within outward. Treated with alcohol and ether, their number becomes less, and they are reduced in size. Hence it appears that they must consist of a fatty substance, which has a very low melting-point, though it is impossible to state anything more definite at this time. The interest which the appearance of these bodies at a low temperature arouses is due less to their presence than to the fact that they occur in the inner lamellæ of the lens, that their number varies in different species of animals, and that their deposit ceases a few months after birth. From this it follows that in the chemical formation of the lens, especially in the older layers of the same, very pronounced changes take place. Since, according to Kuhne and Laptschinsky, the amount of fat is greater in the old than in the young, hence it cannot be due to a quantitative but rather a qualitative change."

These observations may explain why lamellar cataract is formed only in childhood or before birth. Their possible significance in this regard, curiously enough, seems hitherto to have been entirely overlooked. In the hope of throwing further light on the matter, I recently placed the lens removed from a three and a half weeks' old human infant, which had been fixed in formalin, in 1 per cent. osmic acid for five days. The lens was first bisected to insure penetration of the solution. On microscopic examination I found a peripheral zone, thickly studded with blackened droplets. These droplets were readily bleached when treated with a saturated solution of potassium permanganate, followed by oxalic acid, thus proving that they were not ordinary fat,

but that they were probably related to myelin.* If these findings are confirmed by further observations that I hope to make, they will prove conclusively that lamellar cataract is due to the undue persistence of lipoid droplets normally present in fetal lenses. Their persistence in cases of congenital cataract possibly could be explained by a faulty fat metabolism, either dependent on or associated with the faulty calcium metabolism. Ultimately it is possible that they may be replaced by lime salts or other substances.

To explain the probable relation of coralliform cataract to faulty calcium metabolism seems difficult. Professor Osterhout informs me that the conditions under which the proteins crystallize in living tissues are not definitely known, but that almost any protein may be made to crystallize by withdrawing the salts from the solution in which it is dissolved. The possibility suggests itself, therefore, that the crystallization of the lens protein in coralliform cataract may be dependent upon a deficiency of calcium or other salts in the blood or aqueous humor.

As to the time when the crystals are deposited, it would seem that, owing to the softer consistency of the fetal lens, the conditions are more suitable for their deposition in fetal life than after birth. The great abundance and large size of the crystals seem to exclude the possibility that they are precipitated in the fluid contents of the embryonic lens cavity.

Whatever the exact natures of the droplets in lamellar cataract and of the crystals in coralliform cataract may be, it seems sufficiently clear that the lens opacities in each case result from some metabolic disturbance and are not primarily due to degenerative changes in the lens itself. We may, therefore, classify congenital cataracts into two chief types, namely, developmental cataracts, exemplified especially by

* Deutschman,[9] who made the first microscopic examination of a lamellar cataract, assumed, but without proof, that the droplets described by him were of myelin.

posterior polar cataracts, and metabolic cataracts, exemplified by lamellar and coralliform cataracts.

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PERMANENT VASCULAR CHANGES FOLLOWING INJURIES TO THE EYE.

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The cases here reported have these points in common. In each there was a clear and definite history of severe injury to the region of the eye, followed immediately by permanent blindness. The nutrition of the eyeball remained good, and there was no obstacle to ophthalmoscopic study of the late results of the injury. In each case the vascular conditions found were of particular interest.

CASE 1.—M. B., a retired rancher, general health always good, aged seventy-eight years, was struck on the right eye by a chip of wood thirty-nine years ago, and from that time the eye was blind. There was no protracted or severe inflammation. Cataract formed, which was extracted ten or twelve years later, without restoring sight.

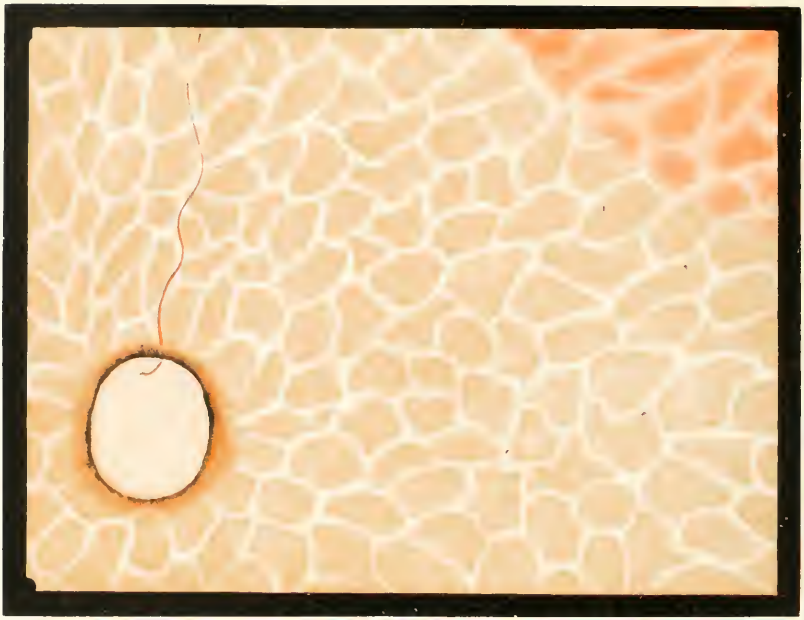

FIG. 1—OBLITERATION OF CHOROIDAL VESSELS (JACKSON)

FIG. 2—AVULSION OF OPTIC NERVE AND RUPTURE OF CHOROID (JACKSON)

December 25, 1915: The left eye had been losing its sight for two or three years. Vision was reduced to light perception, with good light projection, by senile cataract. In each eye there was an old non-vascular pterygium, extending 3.5 mm. on the nasal side of the cornea, which had probably developed before the injury.

The cataract was removed from the left eye by extraction with iridectomy. Healing was uneventful. Corrected vision obtained was 0.7 with a sph. + 12 ⊃ cyl. − 3, axis 80°. The ophthalmoscope showed in this eye a normal fundus.

In the right eye there was an upward iridectomy, the crystalline lens was absent, the remaining capsule almost clear. The vitreous presented a few small opacities. The ophthalmoscopic picture is reproduced in Fig. 1. But one small retinal vessel was visible, passing upward. The optic disc was grayish white and quite devoid of small vessels. Its margin showed a normal ring of pigmentation corresponding to that of the left eye. The whole central region of the choroid, extending from four to six disc diameters from the disc, showed complete absence of retinal pigment and of the choriocapillaris, and obliteration of the larger choroidal vessels. The latter were replaced by grayish-white bands, sharply outlined by retained choroidal pigment. In the periphery of the fundus the capillary circulation and the larger choroidal vessels were normal in appearance, there being very little retinal pigment.

CASE 2.—B. W. G., attorney, aged thirty-six years, good general health. When seven or eight years old was struck in the left eye with a pointed stick and has been unable to see with this eye since. V. R. E. = 0.8, with sph. + 0.62 ⊃ cyl. −1.12 axis 80° = 1. L. E. = 0.012, not improved by glasses. Externally both eyes appear normal, except that the left diverges 50 to 55 centrads.

Ophthalmoscope: Right eye: Media clear, choroid slightly thinned and patchy. Disc slightly red and hazy, with small central physiologic cup.

Left eye: Dust-like opacities were found in the upper temporal part of the vitreous. Other parts of the fundus sharply seen. Retinal pigment layer and choriocapillaris

were completely atrophied except in the upper quadrant of the fundus. Bands of white tissue, made up of strands that overlap each other, run from the optic disc to the extreme periphery, accompanying the upper nasal and temporal vessels; and a third linear band runs horizontally outward. Many of the larger choroidal vessels show the grayish-pinkish-white of choroidal sclerosis. The color is shown in Fig. 1. There were many small pigment deposits in the retina, some in front of the retinal vessels, some branching like bone corpuscles, some rounded, most of them irregular in outline. The optic disc was red and slightly hazy. The retinal vessels were rather small, especially the arteries, except upward, where they were about normal. Above the disc the fundus was 2 D. myopic; in other directions it started with emmetropia at the disc margin, and became gradually more hyperopic, reaching 4 D. at the periphery of the visible fundus. Oblique illumination showed a linear Y-shaped opacity in the posterior nucleus or deep cortex, a fine gray line not visible with the ophthalmoscope.

This eye had gradually diverged to 55 centrads. This was corrected by advancement with tenotomy. The healing in all respects was normal, and vision continues unchanged.

CASE 3.—J. O. E., boy of twelve years, near-sighted since four years of age, was knocked down by a cow three years ago. He was struck in the left eye, which had better vision than his right before that time. The lower lid was torn away except at the nasal end; the upper lid has drooped ever since. The eye was torn out of its socket, but was replaced by the physician who saw him at the time, and the wound stitched.

The left lower lid presents an inconspicuous scar extending 37 mm. down and out from near the everted lower punctum, and having three branches. The eye is about in normal position, but is rotated down until the upper margin of the left cornea is about on a level with the lower margin of the right, when the right eye is in the primary position. The margin of the left orbit feels normal, except the lower margin near the nose, where it is irregular.

There is complete ptosis of the left eye, but the palpebral fissure can be opened to full width with the fingers. It then

shows about 5 mm. of cornea, with the sclera above it. The extreme opening of the fissure by voluntary effort is about 4 mm., showing the upper 2 mm. of the cornea and 2 mm. of sclera. The eye can be turned up 2 or 3 mm., and laterally almost to the normal extent. Movements of the right eye normal. The left eye seems fixed by scar tissue below and back of it.

The pupils in fair light are: Right, 3 mm.; left, 6 mm., in diameter. Movements of the right pupil normal; left a sluggish consensual reaction; no direct reaction to light. Both dilate to 8.5 mm. with homatropin. V. R. E. = 0.12; with sph. $-6 \bigcirc$ cyl. -1 axis $178^\circ = 1.1$; L. E., no light perception, 2 D. myopia. The crystalline lens is in normal position in the left eye.

The ophthalmoscope shows the right eye, media clear, disc normal. Choroid rather light and patchy. Left eye: Media clear; the general appearance of the fundus resembles that of the right eye. The optic disc is replaced by an oval, bluish-white area, vertically about one and one-half times the normal diameter of the disc in length, and the width of the usual disc, with brownish-black pigment splotches all around its margin, which is a little irregular, as shown in Fig. 2. This space is slightly depressed, not over 1 D., and seems to be filled in with connective tissue. It is devoid of vessels. Retinal vessels of about normal size and appearance emerge at the upper and lower ends of this area. To the temporal side of the disc is a crescentic area presenting the usual appearance of so-called rupture of the choroid.

These three cases take added interest when we compare them, especially with regard to the points in which they differ. The striking thing in case 1 is the almost complete disappearance of the retinal and choroidal vessels, with absence of evidences of serious intraocular inflammation. The condition seems best explained by the hypothesis of injury to the vessels back of the eyeball, causing complete thrombotic obstruction in the area of distribution for the posterior ciliary arteries, with preservation of the circulation in the region of the anterior ciliary arteries, as shown by

the circulation, apparently normal in the anterior portion of the uveal tract. Along with the posterior ciliary it is evident that the central retinal vessels had suffered, and both had been obliterated, except the single small branch extending upward from the disc and its visible complementary vessels. We must conceive that from some source a diminished nutritive supply was kept up, sufficient to prevent tissue necrosis. But the anastomotic provision for this was too late or insufficient to reëstablish any of the obstructed vascular trunks.

The picture of choroidal sclerosis, obliteration, or atrophy of the larger choroidal vessels, or disappearance of the choriocapillaris, is well known, and frequently alluded to in the literature; although, curiously, any broad, complete account of the condition seems to be lacking. The allusions to it and descriptions of it are, perhaps, most numerous in Adams' "Ophthalmoscopic Diagnosis." But almost invariably this condition has been confined to quite limited portions of the fundus, and evidently arose in connection with general vascular disease or some local inflammatory condition. No case of such extensive vascular change, with so little evidence of other disease in the eye, following trauma, seems to have been recorded.

Case 2, in contrast with case 1, presented evidence of choroidal sclerosis in small areas, and obliteration of some of the vessels, while others were normal in appearance. The retinal circulation remained practically normal. With these sclerotic changes were distinct evidences of severe inflammatory reaction. The white streaks behind the retinal vessels were such as would be likely to follow extensive hemorrhage; and with them were associated pigment massings and atrophies that pointed to inflammatory reaction. These conditions were widely distributed, but somewhat less pronounced than those shown in the plate accompanying the article by Hepburn.[1] This association of late effects

of hemorrhage and inflammation we have all seen with the ophthalmoscope following trauma; and it is abundantly illustrated in Lagrange's "Atlas d'Ophtalmoscopie de Guerre."

Case 3 must be regarded as one of evulsion of the optic nerve, partial or complete. It would be impossible to determine just how nearly complete it has been.

The reported cases of evulsion of the optic nerve fall naturally into two groups. In a larger number of cases the eyeball has been completely gouged out, or has hung by tissue that was promptly divided; or any view of the interior of the eye has been prevented by massive hemorrhage and subsequent opacity of the media. This case belongs with the smaller group, where valuable ophthalmoscopic observations have been possible, sometimes immediately after the injury, but generally after a longer interval. One of the most interesting of recorded cases is Lang's[2] case, in a boy of fourteen years, seen within twenty-four hours after the injury. V. = 3/60. The upper half of the field of vision was gone, and at the lower part of the optic disc there was a black chasm where the nerve had been torn loose. At the end of six weeks this chasm had filled in, the dark cavern being represented by a shallow white pit 1.5 D. in depth. Vision and the fields remained unchanged.

In the case reported by Salzmann[3] the ophthalmoscope showed at first a deep hole in place of the optic disc, which later seemed to fill from the bottom and contract. Birkhaüser's[4] case, which was also caused by the horn of a cow, was seen the day after the injury. The horizontally oval optic disc was bounded below by a black crescent where a portion of the nerve had been torn loose. At the end of six weeks this was replaced by a white-gray area, showing no parallax as compared with the optic-nerve head. The field of vision remained unchanged. In other cases, first examined oph-

thalmoscopically at a later date, the appearances presented were similar.

Some doubt was raised as to the reality of the evulsion of the optic nerve in case 3 by the comparatively normal appearance of the retinal vessels, although the eyeball had been completely dislocated from the orbit. But this has been noted in the cases where the actual partial tear was seen immediately after the injury. It is quite striking in Lang's representation of the appearance at the end of six weeks. In Birkhaüser's case the upper vessels might be regarded as normal, and the lower vessels appear at the edge of the black chasm, of apparently normal size, only each of the main branches becomes embedded in a large, club-shaped hemorrhage.

In the case reported by Natanson,[5] which was seen two weeks after the accident occurred, with the cavity left by removal of the optic nerve partly filled in, the retinal vessels were not greatly altered in proportion, but appear most of them at the margin of the cavity, as in my case.

In Parsons'[6] case of division of the optic nerve by a stab wound, while there was great edema of the papillomacular region resembling embolism in a retinal vessel, although pressure showed the blood in the vessels to be under reduced vascular pressure, they were of good size, the veins slightly larger than the arteries, and both were of nearly the same color, which resembled that of normal veins.

Parsons suggested: "As soon as organization is sufficient to withstand the low venous pressure the vessels will commence to refill through the direct and the indirect cilioretinal anastomoses near the optic disc. These anastomoses are rather venous than arterial; hence the veins will fill up first. This is what we have seen actually occurs, and it takes place on the fourth or fifth day. The blood is, of course, derived from the recurrent branches of the intact anterior ciliary arteries, and also from any posterior ciliary arteries which

may escape section. The retinal arteries probably fill up first from the periphery, the blood trickling in the wrong direction from the already refilled veins."

Cases in which a histologic examination of the parts was made have been reported by Reis[7] and Liebrecht,[8] but their accounts throw little additional light upon the ophthalmoscopic appearances. The anatomic conditions of recent rupture of the choroid have been observed by Alt.[9]

In every serious injury to the vessels thrombosis is a first step in the process of repair. In the vessels of the eyeball and orbit it rarely extends sufficiently to increase the damage done by trauma. In the majority of cases it helps to secure reëstablishment of the circulation. But it may become a factor in extending the damage done within the eyeball, as has occasionally occurred in the orbit following injection of paraffin into the tissues, or injections in the region of the lacrimal sac. It might be well to consider this even in connection with operative trauma.

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A CASE OF EVULSION OF THE OPTIC NERVE.

CAPTAIN EUGENE M. BLAKE,
M. R. C., U. S. Army.

If one may judge by the paucity of references in the literature, evulsion of the optic nerve is a rare condition. Undoubtedly the nerve is separated from the globe oftener than we realize, but because of other changes which make it impossible to examine the fundus, and because the injury which produces the evulsion may be associated with grave cerebral symptoms and fatal termination, we are not permitted a great familiarity with this form of ocular traumatism. Natanson, writing in 1912, stated that his case was the sixteenth in the literature, and since that time there are but two more cases, so far as I can find. The only complete description of evulsion occurs in the section of Injuries to the Optic Nerve in the Graefe-Saemisch Handbuch, with brief discussion in the American Encyclopedia of Ophthalmology and in Parsons' "Pathology of the Eye."

The terms avulsion and evulsion appear to be used in the same sense by different authors, and it would be well to employ one term consistently, rather than use two for the same condition. Avulsion is derived from the Latin *avellere*, meaning "to pluck off," while evulsion comes from *vellere*, "to pluck," and *e* = "a plucking out." The latter word would seem to be the more descriptive of the two, for that is what actually occurs. The optic nerve is plucked out of the globe at the point where it enters. Salzmann, in 1903, was the first to label the condition, and he called it evulsio optici nervi.

The case which was seen by the writer occurred in a Russian Jew, aged twenty-nine years, on May 14, 1917. Early in the morning on that date, while driving a baker's wagon, he collided with a milk team. His own wagon was overturned, throwing him to the ground in such a manner that his head struck upon a trolley track, his wagon falling on top of him. He was brought into the New Haven Hospital at 7.30 A. M. the day of the accident, and the following notes recorded upon his chart:

Examination of Head and Neck.—Calvarium: Scalp wound over left parietal region near midline, about two inches long. Closed by black silk sutures. No line of fracture or depression felt in skull. Left eye badly lacerated and bulging from orbit, enucleated in the accident room. Right eye, both lids swollen and ecchymotic. Marked conjunctival hemorrhage. Pupil slightly dilated and does not react to light, nor has patient perception of light.

Ears: Negative; no bleeding.

Nose: Compressed and deviated to left side. Loose from its deeper attachments. Can insert fingers into antra.

Mouth: Complete fracture of maxillæ, which are loose from their attachments. Both maxillæ apparently crushed upward and backward. No fracture of mandible detected.

Face: Wound left side chin one inch long. Wounds of mucous membrane, inner side cheeks, especially on the left side, near angle of mouth.

Chest: Fracture of left clavicle about center, dislocated anteriorly. Not much over-riding.

X-ray Report.—Fracture of both maxillæ. Possible fracture right mandible. Fracture into left orbit. Multiple fractures of maxillæ, nasal and orbital bones. Marked displacement of fragments. Possible fracture of coracoid process. Fracture left clavicle and third rib.

The physical examination upon entrance into the hospital is given in detail, as it affords an explanation of the mechanism of production of the evulsion of the optic nerve in this case. The patient was thrown from his wagon, falling upon his head in such a way that the force of the blow was directed upward and to the left. This accounts for the fracture and separation of the superior maxillæ and nasal bones and the extrusion of the left eye from the orbit, as evidenced by the

location of the wounds. Evidently the direct line of force was upward and to the left, but enough pressure was disseminated to the right to produce just sufficient protrusion of the right eye to separate the optic nerve from the globe without rupturing the sclera and causing collapse of the eye, as occurred on the left side. It is as though one pulled an apple from its stem.

The eye-ground was examined for the first time one week after the accident. The pupil was moderately dilated and absolutely immobile. The media were perfectly clear, and the anterior chamber was of normal, or only slightly increased, depth. The region of the papilla was occupied by an excavation, the depth of which was at least 8 D., but was very difficult to estimate, as there were no details to be made out at the bottom of the excavation. In color it was nearly uniformly gray, and one had the sensation of looking into a deep well.

The diameter of the hole was slightly greater than that of the normal disc. The retina was torn on the nasal side of the opening and separated from the choroid for a short distance. There was a large hemorrhage in the upper part of the retina and numerous smaller hemorrhages were scattered about the fundus. A few retinal vessels appeared to have a circulation of blood through them, but the greater part were white and bloodless. Veins and arteries could be differentiated with difficulty.

In the course of a few weeks the excavation at the site of the nerve-head was filled in with new-formed connective tissue, the hemorrhages were absorbed, and the circulation restored in several of the vessels in the lower portion of the retina. The patient made a good recovery in all respects, except, of course, his vision, which was nil. His temperature ranged from 97° upon admission to 101°, reaching normal on the twenty-eighth day. He was later transferred to the State Asylum for the Blind.

Evulsion of the nerve is produced in two different ways: First, and the more commonly reported, by the entrance into the orbit of a foreign body, such as a sharp stick or the point of an umbrella, pieces of metal, stone, or shot. Here the

foreign body impinges upon the nerve, making traction backward and at the same time forcing the globe forward. The pyramidal shape of the orbit and the facility with which the globe can be pushed aside contribute to the production of this form of injury. In the second group the separation of the nerve results through the application of a crushing force, driving the eyeball forward and overextending the optic nerve until it is pulled out of the scleral opening. The case of Reis, where the upper margin of the orbit was comminuted by the kick of a horse's hoof, is a good example, and the subject of the writer's case perhaps especially well exemplifies this type.

Our knowledge of the changes in these cases is slight because the number of cases which have been studied from the beginning and for some time after is small. The separation of the optic nerve manifests itself in the early stage by a complete loss of the papilla and its vessels. At the site of the papilla is found a deep excavation. Birch-Hirschfeld found, in a partial separation after a penetrating wound, an excavation of 6 D. (2 mm.), and Salzmann, after a shot injury, a complete separation with a 12 D. (4 mm.) depression. The absence of the papilla shows as a gray or grayish-blue hole. The retina may be more or less completely torn away, and if the latter, the retinal circulation is lacking. The choroid lies free, raises itself slightly, and the border of the separated retina shows a marked opacity. If the retina is not separated or only in a circumscribed place, then the retinal vessels are completely or entirely retained. The retinal circulation may be greatly increased by the proliferation of capillaries on the nerve-head. Further, hemorrhages at the papillary border in the retina, as well as at the macula, appear. With partial separation, a deep excavation may remain behind or the excavation may be filled in by connective-tissue growth, which piles up in a sheaf-like deposit. Circumscribed pigment accumulation may appear.

As previously mentioned, the separation of the nerve may be partial or complete. Birkhaüser's patient suffered a penetration of the orbit by a cow's horn, and with the ophthalmoscope he saw a small oval nerve-head, with the long axis horizontal. The inferior margin was embraced by a very dark semilune, which took the place of the missing portion of the nerve. There was a marked parallax between the center and the edge, which left no doubt that the nerve had been partially torn loose in the lower quadrant of the scleral canal. The retinal vessels were lacerated in this direction, and in six weeks the semilune had become replaced by new-formed tissue.

The dural sheath is rarely torn, as was demonstrated in the case of Dalen.* Microscopic sections of the eye enucleated because of glaucoma, two and one-half years after the accident, showing a complete separation of the nerve but an intact dural sheath.

Experiments on cadavers were made by Hess in 1907, to produce evulsion of the optic nerve, but with absolutely no success.

It would seem quite likely that in severe head injuries, such as occur in automobile accidents, train wrecks, etc., a more careful examination of the eye-ground or of the enucleated eye would show the evulsion of the optic nerve to be less rare than appears to be the case.

* Ophthalmoscope, vol. viii, p. 519.

OCULAR AFFECTIONS DEPENDENT UPON DISEASE OF THE TONSILS.

(Report of Two Cases.)

HOWARD F. HANSELL, M.D.,
Philadelphia.

Case 1.—Mrs. W., aged thirty-five years, was admitted to the Jefferson Hospital May 20, 1917, under the care of Dr. F. X. Dercum. She stated that in the night of May 16th she went to bed in her usual good health. On Thursday morning, May 17th, she awoke to find the right eye entirely blind and the sight of the left eye diminished. By Saturday she was blind also in this eye. She stated that on Thursday she could see one-half of objects, and this half was obliterated at the expiration of four days.

At the time of my first examination she had no light perception in any part of the field of either eye. The pupils were dilated ad maximum, and did not contract under strong electric light or in association with convergence. Radiographs showed no abnormality of any kind in the bones of the cranium or the contents of the skull. The sinuses of the head and face were clear. The Wassermann test of the blood and the spinal fluid was negative; when the cord was tapped, the fluid was forced out through the canula slowly and showed no sign of increased pressure. It contained no blood-cells or other cells indicative of any general or special etiology. The optic nerves were swollen, and several superficial hemorrhages were seen in the retina adjacent to the disc of the left eye. The boundaries of the nerve-head and the lamina cribrosa were obscured by edema. The edema did not extend far into the retina. The left nerve was swollen slightly more than the right. At this time there were no hemorrhages in the right eye. Examination of the urine, quantity and quality, was negative. The heart and circulatory system and the abdominal organs appeared to be

normal. The patient had no nausea or vomiting; no headache or dizziness; no pain. Indeed, the only complaint was of blindness.

She was of average height, rather fat and flabby. The only recent illness was tonsillitis, from a mild attack of which she recovered four weeks before admission into the hospital. For this affection she had been under the care of a physician.

Report of the examination of the throat by Drs. Chevalier Jackson and Fielding O. Lewis was that the tonsils were cryptic and contained broken-down tissue and probably pus, and ought to be removed. Up to this time the treatment had consisted of daily sweat baths, mercurial inunctions, and potassium iodid. No improvement whatever was apparent. Indeed, while the optic neuritis became no more pronounced, two or three hemorrhages had appeared in the retina adjacent to the nerve in the right eye. The pupils maintained their wide dilatation and light perception remained absent. Twenty-four hours after both tonsils had been enucleated perception of light returned to the left eye, and in one day more to the right eye. The hemorrhages began to be absorbed, and the swelling of the nerve-head, never higher than one diopter, to subside. During the eleven days of blindness no new symptoms developed.

Mercury was badly borne and had to be discontinued. The iodid and the baths were kept up and strychnin was added. Each day after the tonsillotomy vision became better until it equaled counting fingers at six feet. The nerve-heads have begun to grow pale on the temporal sides; the optic neuritis is passing into atrophy. On June 13th daily applications of high-frequency current were commenced. The return of vision has been gradual and not limited to any part of the fields. Simultaneously the pupils began to decrease in size and the reaction to light was reëstablished.

Without following in detail the recovery of vision, it may suffice to state that it was gradual, and that in March, 1918, nine months afterward, it equaled R. 6/10, L. 6/7.1.2. At this time the optic discs were pale and their vascularity was reduced. The fields of vision were not abnormally restricted for white, but slightly and concentrically for colors.

all of which were slowly distinguished. During the first months of recovery she was treated with strychnin, potassium iodid, high-frequency current, and massage.

After eliminating every possible cause by careful and thorough investigation of the other organs of the body, it was concluded that the source of infection was the purulent tonsils. The rapid loss of vision in each eye and the speedy recovery after enucleation of the tonsils seemed to point unquestionably to this etiology.

The onset and cause of the blindness do not correspond to the usual history of acute axial neuritis. In this affection pain in the forehead and temple or deep in orbit is a constant symptom, increased by movements of the eyes or by deep pressure. Vision declines rapidly. The pupils are dilated, and while they do not respond to light, contract consensually. With increasing loss of vision the pain grows less. The blindness is seldom complete and binocular. The fundus remains normal, or there is a moderate degree of optic neuritis. After several days' treatment by sweats or inunctions the periphery of the field is restored, but a large central scotoma persists.

In perineuritis or neuritis interstitialis periphera, the central vision is preserved and the field is concentrically limited for white and colors.

CASE 2.—Mrs. C., aged thirty years, for several weeks had repeated attacks of iridocyclitis. In the intervals the eye cleared and pain subsided. Vision remained dull. For the last five days and nights the pain has been severe and uninterrupted, and she has had but a few hours' sleep. V. = 3/200. The cornea was stippled, anterior chamber deep, and the iris partially attached to the lens-capsule. The ciliary region was excessively sensitive to touch, and all the ciliary vessels were injected. Local and constitutional remedies had been ineffective. She was admitted to the Jefferson Hospital and double tonsillotomy for purulent disease of the tonsils was performed by Dr. Fielding

O. Lewis the same day. In a few hours the pain had entirely disappeared, the synechia were broken off, the pupil round, and the injection decidedly less. In twenty-four hours after the operation she left the hospital practically recovered. Ten days later scarcely a vestige of the inflammation remained. This was a most striking and sudden cessation of the pathologic process, indicating beyond question that the ciliary region was being constantly fed with toxic material from the purulent tonsils.

No bacterial examination in either case was made of the pus taken from the tonsils. The infection is probably not dissimilar to that caused by abscesses around the teeth, for which the staphylococci are usually held responsible.

Steinburger,[1] in discussing the mode of eye infection from a dental focus, which may be identical with that of tonsillar disease, refers to Lelongt's view that it takes place through channels in the bone, subperiosteal tissue, or by way of the lymphatics; Polet's view that, in addition to these, infection may be carried through the nerves; Dutoit adds the ophthalmic vein and cavernous sinuses. He asserts that a direct extension through the venous channels from the diseased area to the cavernous sinuses is anatomically possible and unquestionably has produced a serious thrombosis and its symptoms.

E. V. L. Brown[2] reports a case of sympathetic iridocyclitis and choroiditis with preservation of useful vision in the sympathizing eye. The enucleated left eye showed typical changes. The right eye had thickened, tumefied iris with precipitates. Marked improvement did not occur until infected tonsils were removed, when vision improved from counting fingers to 4/10, and has retained this acuteness for fourteen months.

Other cases, notably those reported by Brown and Irons,[3] to the number of sixteen out of one hundred, point to the same etiology. Zentmayer[4] says "that the tonsils have not more often been found to be the source of the ocular infection

is surprising, when we consider how frequently they are blamed for general infection." The answer to this comment lies in the ignorance and carelessness of the oculist. In view of the great importance of focal infection in ocular disease, to which the attention of the profession was called by de Schweinitz in his masterly paper read before the Ophthalmological Section of the International Medical Congress held in London in 1913, it becomes our plain duty to investigate the condition of the tonsils in every case of intraocular inflammation.

William Lang[5] reports ten cases in private practice; in six there was disease of the tonsils.

I would refer those particularly interested in this subject to the paper of Dunn,[6] of Richmond, Va., who describes a number of cases of uveitis due to tonsillar disease and relieved by treatment directed to the tonsils. Such papers impress us with the necessity of diligent attention to the tonsils in inflammation of the uveal coat that are not venereal, tubercular, or traumatic.

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ACUTE RETROBULBAR NEURITIS OF SEPTIC ORIGIN, RESULTING IN RAPID AND COMPLETE LOSS OF VISION.

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New York.

Although the eye history of this case, previous to the attack of retrobulbar neuritis, has no special bearing on the subject, it is in itself interesting, and I shall report it briefly.

The patient, Mrs. S., first consulted me in 1896; her age then was forty-four years. She is a lady of means, and had, of course, most comfortable surroundings, but never seemed very well, and had a great many things to worry her. At that time she was complaining of severe headaches, which she referred to her eyes. She had always had trouble with her eyes, and was wearing glasses at the time. The refraction for the right eye was sph. +1.25, and for the left eye, sph. −1.25. With these glasses she had 20/20 vision in each eye. I found a hyperphoria of 3 degrees. At that time I questioned that this small amount of hyperphoria was the cause of her eye symptoms. My experience since then has shown me that small degrees of hyperphoria frequently produce asthenopia, and correcting it usually gives relief. I prescribed for her glasses with a prism of 2 degrees; these apparently gave her immediate and perfect relief, which continued for several months, when suddenly a pair of new glasses brought on a return of the headaches and feeling of eye-strain. She submitted these glasses to me at once; and I found that the optician had placed the prism base up instead of down. The correction of this mistake relieved the patient immediately, and she continued to have comfortable use of her eyes for many years, with some modification of her glasses for increasing presbyopia.

On March 27, 1917, the patient consulted me. I had not

seen her for over a year, during which time she had been in rather poor health, suffering with severe pains in her head and neuritis in her neck and arms. She had been under the care of several physicians in Boston; and for some time had been treated by an osteopath there, whom she felt relieved her very much. The eye condition at that time had changed but very little, a slight increase in the amount of hyperphoria, it being 4 degrees. I changed her glasses and advised her to consult her family physician in New York, who had taken care of her off and on for many years. I even then suspected the probability of some sinus trouble. An *x*-ray was made, the report on which is as follows: "A root fragment had been pushed into the right antrum. The antrum also shows a distinct occlusion of its air-space, which possibly may have been caused by the displaced root fragment. The rest of the sinuses appear quite normal. There is considerable constriction in the nares, caused by the deviated septum and enlarged turbinates. The sella is smaller than the average. There is no evidence of any infection about the teeth." The antrum was operated on April 6, 1917, and found to contain a considerable amount of green pus. Permanent drainage was established through the nasal cavity. The final bacteriologic report of cultures from the antrum showed streptococcus viridans. The blood culture proved negative. The patient seemed to be relieved of her headaches subsequently to this operation, although a slight septic temperature continued. She made an uninterrupted recovery as far as the operation was concerned.

On May 7, 1917, she complained of blurring of vision in the left eye. About this time the headaches had returned with some intensity, and the temperature was a little higher —was running between 100° and 101°. On May 8th, the next morning, I saw her, and found the pupil of the left eye moderately dilated, no reaction to light, and not even light perception. An ophthalmoscopic examination showed a moderate papillitis, the arteries small, and sluggish, segmented circulation in the veins. The right eye was unaffected, vision being normal. Dr. Weeks saw the patient on the same day, and agreed with me that there was partial occlusion of the central artery of the retina—probably thrombosis of septic origin.

On May 10th the right eye became affected—a similar ophthalmoscopic picture presented itself in that eye; vision reduced to fingers. Dr. Weeks again saw the patient with me, and we agreed that an immediate operation of the ethmoid cells, which we regarded as the origin of the trouble, should be done. This was in the evening of May 10th; the operation, however, was deferred; and the next day an *x*-ray was made, which gave no evidence of sinus involvement.

On May 12th the patient was seen by Dr. Knapp with me. The vision of the right eye had slightly improved, and we were able to demonstrate a distinct central scotoma for form and color, tenderness on pressure on the globe, and also on movement of the eyeball. We regarded her condition as acute retrobulbar neuritis, and the question of an operation on the ethmoid was again considered, but it was determined, after careful consideration and consultation with two well-known rhinologists, futile. The vision of the right eye failed rapidly, and on May 15th the patient was totally blind. Atrophy of both optic nerves followed.

When the case was first seen, both Dr. Weeks and myself were of the opinion that there was a thrombosis of the central artery of the retina, probably of septic origin. After the right eye became involved and we were able to demonstrate the presence of central scotoma for color and tenderness in the orbit, the diagnosis of an acute retrobulbar neuritis was made, in which diagnosis both Dr. Weeks and Dr. Knapp agreed. Notwithstanding the negative findings of the *x*-ray examination, and that the ocular complications did not manifest themselves for a month after the operation on the antrum, I am inclined to the opinion that this case is one of focal infection, the focus probably being in the posterior ethmoid cells, extending from the diseased antrum. The fact that the left eye was the first to be affected, whereas it was the right antrum that was diseased, is not uncommon. This contralateral visual disturbance in nasal sinus disease has been frequently observed by Onodi, as is stated in

Knapp's "Medical Ophthalmology." Whether the exploration of the ethmoid cells would have been of any benefit, as the process was so acute and so intense, is, of course, extremely doubtful.

This case illustrates to me a very wise remark our distinguished guest, Sir James McKenzie, made in New York, recently: "Let us not be exclusively laboratory physicians."

DISCUSSION OF PAPERS OF DRS. HANSELL AND LAMBERT.

DR. EDWARD A. SHUMWAY, Philadelphia: Focal infection as the cause of organic disease of the eye is becoming an increasingly important factor in our work, whether the infection comes from the tonsils, sinuses, teeth, seminal vesicles, or elsewhere. I desire to place briefly on record two interesting cases:

The first was a woman, aged twenty-eight years, who was sent to me in consultation by Dr. H. P. Masland, of Philadelphia, in September, 1913. The left eye had become painful ten days before, and there was severe pain in the left side of the head. Vision shortly became blurred, and in a few days the eye was totally blind. While not in perfect health, there had been no distinct illness, barring an attack of diarrhea with pain the week preceding the eye attack.

The left eye had no light perception and was painful on movement. The ocular rotations were somewhat limited—outward, upward, downward, and, to a less extent, inward. There was no pupillary response to light. The ophthalmoscopic examination showed optic discs with slightly blurred margins and full veins, but no distinct neuritis. The right eye had 6/5 vision, and normal movement, visual fields, and eye-grounds.

She was admitted to the University Hospital and carefully studied by Dr. Charles A. Fife. No metabolic disturbance was found after most elaborate tests. The urine and blood were normal; the Wassermann test, negative. The nasal sinuses were found free from trouble both by *x*-ray study and by Dr. Ralph Butler on rhinologic examination. A suspicious tooth had its filling withdrawn, but no pus was discovered.

The patient was vigorously sweated and purged and given

bichlorid of mercury at first. Later, inunctions of mercury were employed, iodids and strychnin, and the galvanic current. Nearly four weeks after the first visit she claimed she could see hand movements, but not until thirty-one days had elapsed, or thirty-six days from the time of complete blindness, were there true light perception and return of the pupil reflex. Slowly vision improved until from hand movement perception it reached 6/6. The nerve became moderately atrophic. The field of vision was interesting. Instead of a peripheral field with central scotoma, such as is usually found in acute retrobulbar neuritis, a small central area of vision first returned, as shown by the fields taken at the time. This gradually widened, especially on the temporal side, and later a patch appeared on the nasal side, but was eventually blotted out. On the recovery of vision a left hyperphoria of 8 degrees and an esophoria of 18 degrees were found, showing that there had been an involvement of the nerves controlling the ocular muscles, together with the fifth nerve, at the apex of the orbit. Subsequently she left the city, and was placed in the care of Dr. Corser, of Scranton, who wrote me two-and-a-half years later that vision was still 6/6. I may add that later an attack of uveitis occurred in the other eye, at which time a wisdom-tooth which had been causing trouble was extracted, with immediate improvement.

The second case is that of a man, aged seventy-eight years, whom I saw first May 23, 1918. Nine days before, while in good health, he was seized with severe pain in the top of the head, which later became localized in the left eye. Three days later diplopia appeared, three days later sight failed, and for two days the eye had been blind. There was no light perception or pupil reaction. The eyeball did not move outward beyond the median line, and its movements upward, inward, and downward were greatly restricted; the lid was partially ptosed. The eye-ground was quite normal, the nerve-head was not blurred, nor were the vessels altered. The right eye had normal vision with correcting lenses, and the eye-ground was normal.

A diagnosis was made of periostitis of the apex of the orbit, with involvement of the optic nerve, and the third, fourth, fifth, and sixth nerves, and he was admitted to the Lankenau Hospital for study. The urine and blood examination re-

vealed nothing abnormal. Prostate and vesicles were not the seat of any trouble. Wassermann test was negative. The sinuses were not involved, but *x*-ray examination of the teeth revealed abscesses at the roots of each of five remaining stumps, and these were promptly extracted.

Pain in the head and orbit was pronounced, but at no time was there disturbance of sensation in the face or of the masticating muscles, or involvement of any other cranial nerves. After treatment with mercury and iodid and sweats in an electric cabinet, movement of the eyeball began to improve, and all pain disappeared, but up to date, at his last visit, June 26th, there had been no return of vision, and presumably at his advanced age this cannot be expected.

Both cases represent, therefore, types of acute retro-ocular inflammation of the optic nerve, due to an inflammation at the apex of the orbit. The first is notable because of the long absence of light perception (over five weeks) and ultimate recovery of normal central sight. It is also important, as it bears out Wilbrand and Saenger's contention, that the cases of retro-ocular neuritis should not be spoken of as simply acute retrobulbar neuritis, but that they should be classified in three types:

1. *Neuritis axialis*, with a central scotoma, which may be acute, subacute, or chronic.

2. *Neuritis interstitialis peripherica*, an inflammation commencing usually in the pial sheath and progressing toward the axis. These clinically show a concentric contraction of the field, with good central vision and color perception, or, at times, a sector defect where the process extends only from one side toward the axis, and causes loss of a sector or of an entire half of the field, the rest of the field being free and color perception good. In this class I would place my first case, and probably the second would be found to be of the same type, if vision returns.

3. *Diffuse inflammation* involving the entire cross-section of the nerve, as in acute myelitis. Transition forms occur. They believe that *neuritis retrobulbaris* must disappear from our nomenclature, as it is usually understood as causing central scotoma, but need not, depending upon the portion of the cross-section affected.

Most of these cases have been ascribed to rheumatism or

"catching cold"—"neuritis a frigore"; and many cases have been reported following a chilling, as with periostitis, at the mastoid foramen, and resulting facial paralysis. My feeling, however, is that all such cases should be carefully studied for the presence of focal infections, and these carefully eradicated. I believe, under these conditions, the prognosis will be definitely improved.

But it must be remembered that in all cases of acute retrobulbar neuritis the possibility of a late appearance of multiple sclerosis should be considered. I have already placed on record such a case, which commenced as a unilateral neuritis, due apparently to ethmoid sinus empyema; and, with Dr. William G. Spiller, am studying a similar case at present in which the teeth were at fault. I would like again, in this connection, to voice my feeling that multiple sclerosis, in which we have multiple inflammatory foci scattered through the central nervous system, may be originated in a focal infection in the teeth or sinuses, a view which has been corroborated by Stark, in Texas, and by Billings and Rosenau, of Chicago. Particularly if, as Uhthoff has shown, the retrobulbar neuritis be unilateral, and the central scotoma be relative and not absolute, should the diagnosis of beginning multiple sclerosis be suspected.

DR. J. F. SHOEMAKER, St. Louis, Mo.: The question of the manner in which sinusitis causes scotoma, which is one of the more prominent symptoms of retrobulbar neuritis, is of considerable interest. Birch-Hirschfeld believes that it results from the toxic action of the inflammatory products on the nerve-fibers. He believes that the papillomacular bundle is first involved because of its vulnerability, and that this is one of the first symptoms of involvement of the optic nerve in sinus infection. Wilbrand also believes the fibers of the macula are more vulnerable to toxins; that their medullary sheaths are thinner and finer than those of other fibers; and that their axial position makes it impossible for them to eliminate waste substances, such as functional metabolism products or toxins absorbed, as easily as the other part of the optic nerve. We must not forget the possibility of a direct extension of the inflammation to the nerve-fibers by way of a perineuritis. This would cause an interstitial

neuritis and affect the vision; but in such cases we should expect to find a peripheral concentric contraction of the field of vision, which, indeed, has been observed in sinus involvement, so that it is not likely that central scotoma comes about in that way. Compression of the retinal vein at its exit from the nerve-trunk has been suggested as an explanation of this condition, and a case of this kind has been recorded as occurring in a patient in whom there was a tumor of the sphenoid. The changes in the optic nerve-fibers in this case were very similar, on microscopic examination, to those observed in intoxication caused experimentally by methyl alcohol and filix-mas. In Dr. Lambert's case the cause of loss of vision seemed to be due to interference with retinal circulation.

I desire to report a case of large central scotoma caused by hyperplastic sphenoiditis, in a man thirty years of age, in which I believe the affection of the optic nerve was brought about by the action of the toxins on the nerve-fibers. The patient stated that three weeks previously his vision had suddenly become so dim that he could get around with difficulty. I elicited the information that while he had not noticed a marked diminution in vision previous to this, he had been rejected by the Medical Examiner of the army six months before because of his eyes. V. R. E. = 12/100; L. E. = 3/200. The ophthalmoscope showed clear media, but low-grade inflammation of the optic nerves in both eyes. The fields showed some concentric contraction. Color perception in the right eye was limited to five degrees from the point of fixation. In the left eye the color perception was indefinite, so that he was never certain about any color, and there was a marked central scotoma. He stated that he had never used tobacco or alcohol to any extent. As he was a salesman, it did not seem possible that he had inhaled or otherwise absorbed any toxic substance that could affect the nerves. He was referred to a rhinologist, who reported that he had a marked form of hyperplastic sphenoiditis. The Wassermann of the blood was negative, and also the test of the spinal fluid. The rhinologist operated on the left side, but no improvement of vision followed. He was treated by the rhinologist, but in the course of a week or two vision began to get worse in the right eye, and he developed a cen-

tral scotoma of that eye. Four weeks from the first operation the right sphenoid was operated. Following this there was considerable diminution of vision above what had taken place before. He had but 3/200 or 4/200 vision, with a large central scotoma in each eye, and no perception of color in any part of the fields.

A month or so after the second operation he thought that his vision was getting considerably better; but a test showed that he had only 5/200 eccentrically. However, the central scotomas were considerably diminished, and whereas, at the worst of the condition, they had included 15 to 25 degrees from the point of fixation, they were now narrowed down very much; but they still took in the central part of the field, so that his vision, five months after the first visit, was only about 5/200 or 6/200 and the optic nerves were markedly atrophied.

I believe that in this case the action on the optic nerves of the poison generated in the hyperplastic sphenoiditis was responsible for these large central scotomas.

DR. JOHN E. WEEKS, New York City: I want to speak regarding the case that Dr. Lambert has reported. The optic nerve on the left side, when I first saw this patient, was slightly pale, the outlines a little indistinct; the arteries, small; and the veins showing a segmented circulation, with a history of pus having been found in the antrum. At a second examination the arteries on the left side had not increased in size. The veins were still small, but the segmentation had, I think, disappeared. The sudden loss of vision with this appearance of the background was, to me, a picture of interruption of the circulation in the central artery of the retina. The question was whether it was simply from pressure, from spasm, or from a thrombus; to me, the most plausible explanation was a thrombotic condition due to the absorption of toxins. I recommended at that time a free opening of the sinuses, so far as possible, to insure free drainage, as it seemed to me very evident that the condition was due to a septic process. When I saw the second eye involved, the appearances were almost similar to those of the first. The retrobulbar inflammatory condition—term it neuritis, if you wish—was undoubtedly present, but there was ob-

struction to the circulation on the right side, and I was very insistent in my advice to make the drainage from the accessory sinuses free, but was over-ruled. Dr. Lambert fully concurred, I think, in my suggestions in regard to that. The outcome, as you know, was one of complete blindness.

I have at present under observation a case in which the vision was reduced to 6/60, apparently from a sphenoid sinusitis. The vision at the present time, which is three weeks since I first saw the case, is 20/20 minus, the improvement in vision apparently being due to effecting free drainage of the sphenoid sinus on the right side.

DR. G. ORAM RING, Philadelphia: If emphasis was needed on the point of the importance of the study of the throat in such serious cases as Dr. Hansell's, certainly his striking, not to say dramatic, results afford it; but the study of these conditions, while important, does not always yield such notable results as those reported in these cases. Eight weeks ago a student in the University of Pennsylvania, twenty years of age, splendidly developed, came to my office telling me that he was in the sophomore year and was doing well in his work, but was supplementing his regular studies with a military course. Up to two weeks before he had been regarded as a crack shot in his class. Then he began to fall down, so far as the accuracy of his shooting was concerned, and concluded that it was due to blurring of the right eye. Vision in left eye was normal, but his right had dropped to 20/40. Examination showed the anterior part of the uvea normal. There was no exudate nor hemorrhage, but a perfectly definite punctate hyalitis. He was referred to a physician, who reported his condition negative, and I began a series of exhaustive studies to try to find the etiology of the condition. It was found that there were two possible sources of trouble—one in the intestinal tract and the other in the tonsils. The former showed two definite poisons—one an excessive indicanuria and the second a definite urorosein excess. He had never complained of his tonsils, nor did he ever remember an attack of tonsillitis. Examination showed half a dozen crypts that were visible and that evidently had cheesy exudative material in them. The intestinal condition was promptly treated with high enemas and laxatives and with

suitable dietary regulation. Iodid and thyroid were given internally, and each crypt that was available was carefully and repeatedly cleansed with iodin and alcohol. The improvement from 20/40 to 20/20, partly, was noted at the end of four or five weeks. The fundus had not cleared, but was definitely improving. Then came a lull; beyond that condition, for two or three weeks, there was no improvement that I could observe. It then occurred to me that a more accurate investigation of the condition of the tonsil should be made. I simply separated the tonsils from their anterior pillars with the Makuen knives, which immediately showed the presence of a considerable amount of cheesy material. Both tonsils were removed a few days later, with an almost immediate clearing of the vitreous.

DR. T. Y. SUTPHEN, Newark, N. J.: In connection with this discussion I wish briefly to relate one case. In January last an Italian, aged twenty-eight years, came to our clinic with a severe optic neuro-retinitis of the left side. His vision in that eye was almost nil, while the right eye was healthy, with vision of 20/20. He had been under treatment elsewhere for several weeks for syphilitic disease, the Wassermann test being positive. Upon admission radiographs were taken of his head, which showed trouble in the left ethmoid and apparently the sphenoid. Wassermann test was again taken, and found negative. A few days later the ethmoid was cleaned out by an operation. At once the neuritis began to subside and the man made a rapid recovery with 20/20 vision. Although the case was one of specific source, it was added proof of the value of surgical interference.

DR. HIRAM WOODS, Baltimore, Md.: The discussion has been allowed sufficient breadth to justify me, I think, in giving the subsequent clinical history of a case reported to this Society two or three years ago. The paper dealt with eye manifestations of "Intestinal Toxemias." Among the cases was that of a lawyer with double uveitis, involving almost the entire tract. All the clinical and laboratory investigations for the cause were negative except—(1) a suspicion in regard to the right antrum; and (2) indicanuria and other evidences of intestinal putrefaction. While this

intestinal disorder was being treated, the teeth were studied. At the root of a molar tooth there was found a suspicious appearance, and the tooth was extracted. As soon as the tissue was opened up by the extraction, the man went suddenly blind. He had to be led to the hospital. The uveitis was so dense that the fundus could not be seen. After ordinary treatment with sweats, etc., for three or four days it began to clear up.

That brings us up to the time when the paper was written. He then had good practical vision—about 20/40. He subsequently had a relapse, and the right antrum was opened. It contained pus. The left was found normal in *x*-ray examination. He continued to get no better and was sent on to Philadelphia to Dr. de Schweinitz. A rhinologist found the left antrum involved, and that was drained. He was there nearly a month, and came back in a somewhat improved condition. Since then he has had a relapse of the old intestinal condition and appendicitis. Appendectomy was done while the eyes were inflamed and an almost immediate improvement in vision followed. Both antra have been drained, several teeth extracted, an ethmoid operation done, and finally a mastoid operation. He stood them all and now has 20/20 vision in each eye.

This case ran over a series of years, with the discovery of one infection after another. It brings up an interesting question: When have we got down to the bottom of a case? In the presence of a number of sources of infection, how can we tell which is which? Take the teeth: I saw a young woman the other day, twenty-seven years old, with a low grade of iritis, deposits on the cornea, etc. She had had every tooth in her upper jaw pulled out, and the eye was no better. Every tooth was gone, not because there was a positive diagnosis of proved relation between the tooth and the eye condition, but because there was an association of an eye condition with an infected tooth. I think that it is up to us to get down to a little better diagnosis than mere association. The man illustrated a great many sources; and this woman, a perfectly useless sacrifice of teeth, because the *x*-ray picture showed, or they thought it did, certain shadows. It may be justified. I am not saying anything against the theory of infection from these foci, but I am pleading for a little more

accurate diagnosis of cause and effect. At present, we can judge only by results.

DR. WALTER L. PYLE, Philadelphia: I should like to ask Dr. Woods whether the teeth pulled out on *x*-ray suspicion were sound.

DR. WOODS: The patient told me that they were. I did not see them. The dentist had extracted these teeth under protest, on the *x*-ray findings, in a desperate effort to find the cause of the eye condition.

DR. J. T. CARPENTER, Philadelphia: In these days of intensive laboratory research it is rather satisfactory to be able to point to the clinical solution of a problem. Five or six years ago I reported a case before the College of Physicians exactly similar to those reported by Dr. Hansell. A young woman of twenty years was referred to me on account of blindness. There was a moderate neuro-retinitis, with a few scattered hemorrhages and swelling of the disc. Vision was reduced to fingers, and the usual search was made for the cause. There was a marked involvement of the dental region, and she was treated by a specially prepared serum without improvement. She had all the active measures usually adopted for the reduction of the ocular condition. It was only when Dr. Francis Packard removed the tonsil on the side on which vision was affected that a prompt and immediate return to normal vision occurred, and there was prompt subsidence of her ophthalmoscopic signs of neuro-retinitis. She has since enjoyed absolutely normal vision. We can say, in this case, that the tonsillar infection was responsible for the eye condition, because it immediately subsided, whereas for several weeks every other form of treatment had been used without result.

DR. WILLIAM TARUN, Baltimore, Md.: There is this important point that I want to bring out with regard to the teeth: Because there is an abscess, and the dentist extracts the tooth and finds it free, is no proof that the abscess is not present. If the *x*-ray were taken afterward, very likely the abscess would still be seen on the film. In all instances the dentist should curet the alveolar processes, otherwise the sac remains after the extraction is done. That is a point fre-

quently overlooked, particularly where there are abscesses at the roots of the cavities. The toxin comes from a localized abscess, either in the teeth, the tonsils, or elsewhere.

DR. W. GORDON M. BYERS, Montreal, Can.: One thing that stands out clearly in this work during the past few years is that one frequently has in the system multiple foci of infection; and for this reason it will not do, in these cases, to draw from the history a few preconceived etiologic possibilities and rest satisfied with a restricted investigation. We must adopt a standardized type of examination to get the best results and to avoid the evil of which Dr. Woods has spoken. Certain things must be gone through with as a routine. With full information regarding all the etiologic possibilities underlying any given case, we can come to some definite conclusion as to what is the right thing to do.

DR. WILLIAM H. WILDER, Chicago, Ill.: I am glad that Dr. Byers has said what he did. The whole subject is more or less obscure, but there is one element in these cases that must not be lost sight of and which is of great importance, and that is the element of time. We are dealing with a possible inflammation of a nerve structure, and time is important. The earlier we make the diagnosis, and the earlier we discover the focus of infection, if there is such a focus, the better for the patient. We have all seen cases in which blindness came on rapidly a considerable time before there was any sign that could be observed with the ophthalmoscope. This rapidly occurring blindness has been observed in syphilitic leptomeningitis, in cases of ethmoid infection, of sphenoid infection, and of infection from other foci; so that, as a routine measure, we should go through these various investigations to determine the focus of infection as thoroughly and quickly as possible. In all my cases, where there is a suspicious origin, in addition to other examinations, I have the prostate stripped to make sure that there is no latent gonorrheal infection. Whether the lesion in the nerve is a perineuritis, an interstitial or a diffuse inflammation; whether it involves the nerve as far back as the chiasm or affects that portion of it that lies in the orbit, is not always possible to say until we have an opportunity to make a histologic examination. Nor can we tell what the virulence of the infec-

tion is. Infections have varying degrees of virulence, and in some we may expect to find rapid disorganization of this nerve tissue that is so important to the eye. Hence the importance of making such investigations promptly in order to institute whatever method of therapeusis is indicated as soon as possible.

DR. WALTER E. LAMBERT, New York City: I should like to report in connection with Dr. Hansell's paper a case almost identical with the second one reported by him. It was that of a lady with a low-grade uveitis. The Wassermann was negative. There was a history of rheumatism, but she was in pretty good health otherwise. Both tonsils were diseased, and were regarded by exclusion as the cause of the uveitis. These were removed and she made a perfect recovery.

CONCERNING THE ETIOLOGY OF HEREDITARY OPTIC NERVE ATROPHY. REPORT OF TWO CASES WITH INTERESTING *X*-RAY FINDINGS.

WILLIAM ZENTMAYER, M.D.,
Philadelphia, Pa.

In this paper we are concerned only with the etiology of hereditary optic nerve atrophy. The cases here reported present nothing that is new in the symptomatology of the disease, and, as this phase of the subject is thoroughly digested in the monumental work of Wilbrand and Saenger, no essential purpose would be served by restating it.

The opportunity was recently afforded to make an *x*-ray study of the several members of two families in which the disease had made its appearance, and the results of this investigation are the feature of this communication. The findings are in no way conclusive, but are sufficiently uniform to merit attention.

In the first family (C.) there were four children—three

boys and one girl. One boy died of typhoid fever; the other two are affected. The girl is living and unaffected.

CASE 1.—J. C., the first male, who was a stove molder, was affected when thirty years of age. He had the usual children's diseases, but suffered no permanent sequelæ and has been in good health since. Smokes moderately and uses but little alcohol. In February, 1912, he accidentally discovered that the vision of the left eye was poor, although he could see only large objects. The vision of the right eye was seemingly unaffected. By the latter part of August of the same year he had to give up work because of the poor vision in the left eye. Soon after this a blind spot appeared to the outer side of fixation and then spread to the center. He was much annoyed by a continued shower of snowflakes, which was finally replaced by a fog. Never had diplopia. When examined in November, 1917, eccentric V. R. E. = $\frac{1}{180}$; L. E. = $\frac{1}{240}$. Iridic reflexes sluggish. There was an absolute central scotoma about 20 degrees in extent. The optic nerves were greenish gray, and the vessels contracted. The neurologic examination was made by Dr. W. G. Spiller and was negative, except that the tendon-jerks were overprompt.

The nasal examination made by Dr. J. G. Schwerin showed: Nasal cavities negative, tonsils and pharynx red and swollen. The *x*-ray examination made by Dr. Henry K. Pancoast showed an enlarged pituitary fossa both by deepening and in the anteroposterior direction, 13 mm. anteroposterior by 12 mm. depth.

CASE 2.—C. C., the second male affected, first seen in October, 1917, was twenty-nine years of age. Until the summer of 1917 he had been employed in a glass works, the last seven years as a blower. During the summer, three weeks previous to coming to Wills Hospital for failing vision, he did outdoor iron painting. The paint contained Japan dryer. He had, however, no toxic symptoms. Three weeks after quitting this work he noticed that persons seen across a street looked blurred. Vision continued to fail until, by the following week, he could no longer read ordinary print. He thinks the sight of the left eye first began to fail, but of this he is not certain. He was in good health until the spring of 1917, when he had congestion of the lung with pleurisy.

He has smoked about one ounce of tobacco a day for several years. Does not use alcohol. He is married and has one child, living and well. His build is slight and he appears anemic.

Pupils 4 mm. Iridic reaction normal. Interpupillary distance, 71 mm. The upper nasal borders of the papillæ are obscured and prominent. V. R. E. $=\frac{20}{200}$; L. E. $=\frac{15}{200}$. Visual field for right eye shows a scotoma for red extending about 8 degrees on either side of fixation and 20 degrees below and breaking through the limit for red above. At fixation there is an absolute scotoma about 5 degrees in extent (Fig. 1).

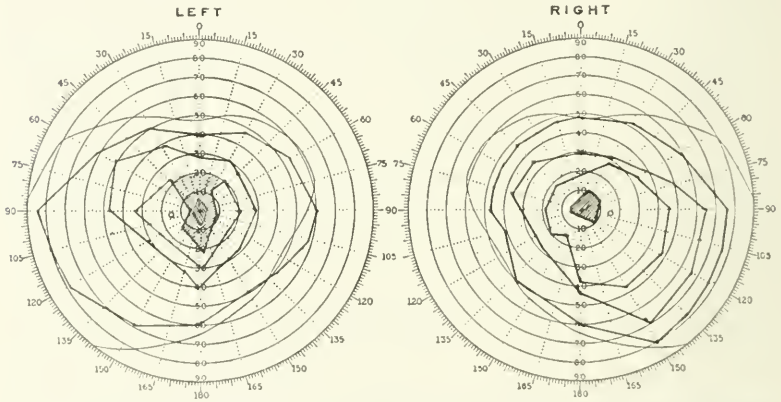


Fig. 1.—Case 2. Fields for form and blue and red. Right eye, relative and absolute scotoma; left eye, relative scotoma.

In the field of the left eye there is a relative central scotoma of about 5 degrees extent. The form fields showed only slight contraction. It was thought that the condition might be due to an intranasal lesion, and this was apparently confirmed by Dr. J. G. Schwerin, who found both nasal cavities full of polypi and a purulent ethmoiditis. Complete eradication of these conditions, however, failed to arrest the progressive loss of vision, but the edema of the papilla disappeared. This was followed by an increasing atrophy.

A further search for the cause brought out the fact that a brother had lost his sight from optic atrophy (Case 1). The

neurologic examination was negative. The aural examination by Dr. Lewis Fisher showed a hyperactive response in nystagmus and diminished responses to vertigo and past pointing, indicating a central disturbance. The *x*-ray examination showed possibly a slight deepening of the pituitary fossa—on the border-line of normal size, anteroposterior 10 mm., depth 11 mm.

The second family (S.) in which this disease appeared consisted of nine children—five males and four females. Of these the fifth and seventh child, both males, are definitely affected; the third, a female, presents suggestive incipient symptoms of pituitary disease. The mother of this family was an only child. She is living at the age of fifty-three years. The father is fifty-three years of age and has normal eyes. There is no consanguinity in the ancestry, so far as could be determined.

CASE 3.—The first member affected (F. S.) is a male, now twenty-six years of age. At the age of twelve years the sight began to fail, and in six months' time he was no longer able to read. He thinks the left eye failed the faster. He was examined early in the attack by a competent ophthalmologist when a diagnosis of optic atrophy was made. Vision has remained stationary in the last thirteen years. Except for scarlet fever at seven, he has had no illness. At school he was a normal pupil until his sight failed.

Examined February, 1918; eccentric V. R. E. $=\frac{1}{1\,2\,0}$; L. E. $=\frac{1}{1\,2\,0}$; pupils equal, consensually, right = 5.5 mm.; left, 5 mm.; right eye slightly divergent. The face is asymmetric—the right side recedes. The skull is high and narrow. The field of the right is contracted to within 40 degrees of fixation, and there is a central absolute scotoma 20 degrees in extent; the left is moderately contracted, and there is a central triangular absolute scotoma 60 degrees by 15 degrees. No color perception (Fig. 2). Both optic papillæ atrophic, with sharply defined laminæ. The vessels are normal in the right and slightly contracted in the left. The arteries are too bright. Under ophthalmoscopic examination the eyeballs develop a jerky movement, usually up and to the right.

The physical and serologic examinations are negative. The *x*-ray examination shows a large pituitary fossa on the border-line of normal—12 mm. anteroposterior diameter and 10 mm. deep.

CASE 4.—The second male affected (E. S.) is twenty-two years of age, a chocolate maker for the past nine years. Two years ago he first noticed that colors did not appear normal, and examination of the eyes at that time is said to have shown an inflammation of the optic nerves. One year later he had repeated Wassermann tests, all of which were negative. These results were confirmed by our examination.

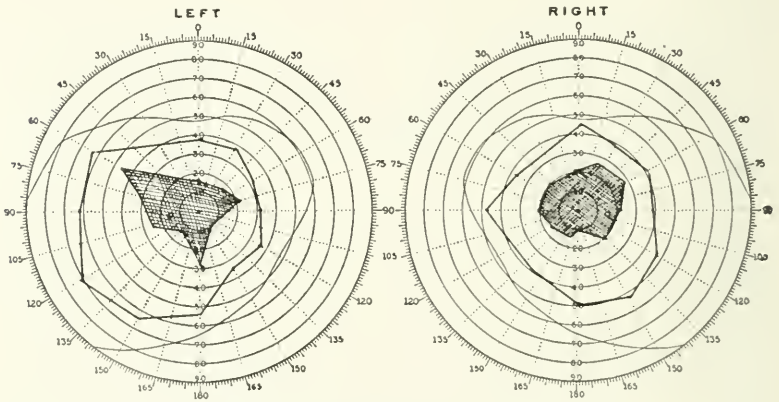


Fig. 2.—Case 3. Form field. Absolute scotoma.

The nose, throat, and ears are normal. The Bárány test is normal. V. R. E. $= \frac{1}{60}$; L. E. $= \frac{2.5}{60}$.

R. E.: The optic papilla, which is atrophic and of a greenish-white hue, has a deep, pit-like excavation. The perivascular lymph-sheaths are full. The vessels are contracted and the arteries too bright.

L. E.: The papilla is gray and the vessels of a better size than in the right eye. The *x*-ray measurements of the pituitary fossa are: anteroposterior diameter, 13 mm.; depth, 12 mm.

The opinion of Dr. Pancoast that in cases 1, 2, and 4 the pituitary fossa was either above the average normal or at the

maximum normal was confirmed when the cases were shown before the Philadelphia Roentgen Ray Society, and it was suggested that other members of the family who were apparently unaffected be examined as controls. Accordingly, one male and one female of the second family were *x*-rayed. The male (C. S.) was twenty years of age, and showed no ocular disturbance other than a high hyperopia. The pituitary fossa measured 9 mm. anteroposterior diameter and 9 mm. deep. The fossa was therefore much smaller than in the two affected members.

The apparently unaffected female (M. S. E.) is thirty years of age. The fossa measured 12 mm. anteroposterior and 10 mm. deep and is, therefore, on the border-line of being abnormally large. She subsequently came to my office for an ocular examination, being fearful of a fate like that of her brothers. She is married and has two living girls, aged eight and nine years. The last two children were boys—the first dead born, the second living but eight hours. She wore glasses at fourteen years of age for a period of one year. She complains of momentary obscuration of vision, a great deal of headache, and annoying sweating of the left side of the head. Corrected V. $= \frac{5}{4}$ (normal).

The right papilla is normal; the left shows all but outer border obscured and slightly prominent. Central lymph-sheath full. The visual fields showed decided concentric contraction for form and color, with a minute central relative scotoma in the right field and an equally small paracentral relative scotoma in the left. Upon a second testing, five days later, on a Peter campimeter, no scotoma could be demonstrated in the left field. She was examined neurologically by T. H. Weisenburg, who found nothing abnormal aside from the left-sided head sweating.

Report of the X-ray Examination Made by Dr. Henry K. Pancoast.

Family C.—C. C.—At the first examination of this patient it was thought that the pituitary fossa was negative, but after an examination of his brother, a reëxamination convinced us that there was possibly a slight deepening of the pituitary fossa. This is about on the border-line of normal size.

J. C.—The pituitary fossa of this patient is slightly past the border-line of normal size, and it is undoubtedly somewhat enlarged, both by deepening and in the anteroposterior direction.

Family S.—E. S.—The measurements of the pituitary fossa showed the fossa to be 13 mm. in the greatest anteroposterior diameter and 12 mm. deep.

F. S.—The pituitary fossa is on the border line of normal. There is no evidence of any unusual pressure. The floor and posterior processes are thin, but not out of proportion to the rest of the skull, although thinner than in the case of the brother, C. S. The skulls of both C. S. and E. S. are of about the same thickness. The left anterior clinoid process is hollowed out beneath, while the right is not. Both are normal, but we usually do not find this combination. Measurements of the pituitary fossa—12 mm. anteroposterior diameter and 10 mm. deep.

C. S.—(One of the normal members of the family.) The skull is thin. The pituitary fossa of normal size, and the processes about of normal thickness and apparently thicker than the two affected brothers. Measurements of the fossa —9 mm. anteroposterior diameter and 9 mm. deep.

Mrs. M. S. E.—(Indefinite pituitary symptoms.) The fossa in this case is large and on the border-line of being normal, as in the case of F. S. The posterior clinoid processes and the floor are thinner than in the normal brother. The skull is thicker than in any of the three brothers. Measurements of the fossa—12 mm. anteroposterior diameter and 10 mm. deep.

Dr. Pancoast concludes that as each of the four cases

Case 1.

Case 2.

Case 3.

Case 4.

Case 5.

Case 6.

Fig. 3.—Cases 1, 2, 3, and 4 affected. Case 5, unaffected. Case 6, unaffected, but presenting indefinite pituitary symptoms.

showed a pituitary fossa which was either at the border-line of being abnormal in size or was slightly past this point, it would seem as though a pituitary enlargement might be regarded as a possible cause of the condition.

Concerning the *x*-ray findings in the female, who certainly has not, now, an optic atrophy, but who has subjective symptoms, ocular fundi, and field changes suggestive of chiasmal disturbance, it would seem not unfair to hold that at the present time it cannot be said to refute Dr. Pancoast's views.

It may be recalled that recently Fisher brought forward the following facts, from which he argues an inherited temporary disorder of the pituitary body as the primary cause of Leber's disease.

Pollock (Amer. Jour. Ophth., August, 1918) reports two cases of Leber's disease in which the radiographs were identical—a shadow like a very small bean, with its concavity downward, situated in but a little below the center of the sella turcica.

Leber noticed a great tendency for the visual defects to appear at or about the age of puberty, and that evidence of a neuropathic type was afforded by such symptoms as headache, vertigo, tremors, numbness of all the limbs, or even epileptic fits; that in several reported cases there were similar symmetric field defects; that patients with Leber's disease, as also those with rapidly developing pituitary body growths, often complain of subjective phenomena of light and colors, often as "seen through a blue mist"; that variation in the degree of central amblyopia which occurred in Leber's disease is more consistent with an outside influence on the visual pathways than with primary changes in the nerve-fibers; that in both conditions there is an epochal relation between the onset and the period of puberty and the climacteric, and that in both, frequently in early stages of the disease, a very mild papillitis can be detected. In one of two affected children of a family roentgenoscopy of the

skull was negative, while the other showed a cellular or honeycombed shadow in the depression of the sella turcica. That similar changes were not found in the two cases he attributes to the fact that the negative finding was in a case of two years' standing. As the visual symptoms in Leber's disease are progressive up to a certain point and then come to rest, he argues that if the lesion which gives rise to these symptoms is due to some disorder of the pituitary body, it also must needs be temporary and transient.

In regards to heredity, Harvey Cushing remarks that there may be certain inherited deviation which may in all likelihood be attributable to transmissible ductless gland properties; and a functional glandular instability may exist in these individuals which makes them more susceptible, under stress, to alterations which border on the pathologic.

It would seem to me that our data are insufficient upon which to draw conclusions. Aside from the fact that the normal fossa must vary greatly in size and often overstep the limits set as maximum normal, our present knowledge of heredity in connection with disorders of the pituitary body is too vague to permit of assertive statements.

I wish to express my thanks to Dr. W. D. Barry for his kindness in permitting me to publish the notes of the second family.

TUBERCULIN IN DISEASES OF THE EYE.

JOHN E. WEEKS, M.D.,
New York.

This paper is written principally for the purpose of exciting a discussion of the questions raised, namely: First, In what condition of the eye and its adnexa is the use of tuberculin indicated? Second, What form of tuberculin can be used to best advantage in diagnosis and in treatment?

Third, In what dose and for how long a period should tuberculin be used in the treatment of affections of the eye?

In establishing a diagnosis of a tubercular process in the eyes, the subcutaneous injection of old tuberculin, "T. O.," is the procedure now generally considered to be the best. Trudeau has found that the subcutaneous injection of 9 mg. of old tuberculin will produce a rise of temperature in a healthy adult; hence it is necessary to employ a smaller dose in conducting this test. In my experience it has been perfectly satisfactory to begin the test with 1 mg. in the adult, ½ mg. in children. (The temperature of the patient should be taken twice in twenty-four hours for one or two days before giving the injection, and the injection given only if the temperature is normal.) The injection is repeated if no local reaction (at the site of the inflammatory process in the eye) is obtained forty-eight to seventy-two hours later, provided the patient's temperature has remained below normal for the preceding twenty-four hours, using 2 mg., and again in forty-eight to seventy-two hours, using 3 mg., if a satisfactory reaction is not obtained by the smaller dose. It may be justifiable in certain instances to use a larger dose, as in some cases of small tubercular foci the smaller dose is not sufficient to excite the reaction. The local reaction in conjunctival tuberculosis is indicated by an increase in hyperemia at the site of the lesion; in scleral and corneal lesions by increase in pericorneal injection, the conjunctiva nearest the lesion being most affected, slight increase in the density of the tissues at the lesion, and in slight cloudiness of immediately adjacent cornea; in iris tuberculosis an apparent exacerbation of the process, manifest by increase of hyperemia at the base, slight enlargement of the tuberculous masses, and increased haziness of aqueous. If the white, fluffy, "mutton-fat" exudation is present, this may be slightly increased in amount. Some increase in pericorneal injection and slight pain referable to the eye may be produced.

The same changes may also occur in ciliary tubercular processes. In tuberculosis of the choroid the haziness over the affected area may be increased, the area involved slightly enlarged, and minute, grayish, punctate patches appear at the margin of the lesion, and vision become more impaired. In tuberculosis of the retina and optic-nerve head similar changes may be produced. In order to observe these changes the eyes must be examined from time to time from twelve to thirty-six hours after the injection, and the condition compared with that preceding the injection. The reaction in the lighter cases may subside in six or eight hours, and in the severe cases seldom lasts more than forty-eight hours. The condition of the eye, after subsidence of the reaction, is usually better than before the tuberculin was given. In no case has the eye been made worse, in my experience. In some cases the local reaction is extremely slight, and in some cases of undoubted tuberculosis no local reaction can be detected.

It must be borne in mind that it is not impossible for tuberculosis and syphilis to reside in the individual at the same time, and that the one process may modify the other, so that a nodular iritis or a disseminate choroiditis may be influenced by both diseases. The writer has seen two such cases affecting the iris. It is, therefore, desirable in many of the cases to make the diagnostic tests for syphilis as well as for tuberculosis, and in the cases in which both are positive to institute treatment for both at the same time. In fact, it is the practice of the writer to give Hg and K. I. in moderate dose in very many of the cases in which tuberculosis of the eye exists, as well as to employ general tonic measures in the broad sense of the term. This brings us to the question, in what cases of eye lesions shall tuberculin be used?

When the writing of this article first occurred to me, it was my purpose to inquire whether tuberculins were of any

service in the treatment of any disease proces
those excited by the tubercle bacilli. This oc
because of the analogous use of diphtheria ant
I found in 1913 was being employed in Glasgow by Dr. Maitland Ramsey in ulcer of the cornea of various kinds, and in some cases of phlyctenular keratitis, with alleged beneficial results; also that it was being used in one of the eye clinics of Vienna in similar affections of the eyes. The rationale for the practice was not very definite, but was based on the supposition that antibodies were introduced in the system which were antagonistic to the agents that were concerned in the production and continuation of ulcers of the cornea and other eye affections in which it was used. Although the results obtained by those who used the antitoxin were said to be very good in many of the cases, I have not considered it advisable to adopt the practice. In my reading of literature relating to the use of tuberculin I have not found mention of the use of this remedy for the treatment of any but tuberculous lesions. I raise this question in order to obtain an expression regarding it on the part of the members present. It is quite evident that tuberculin should be employed, first, in all cases of diseases of the eye and its adnexa in which a local reaction is excited by the subcutaneous introduction of test doses of tuberculin; second, in those cases of suspected tuberculosis—(*a*) in which a general reaction to tuberculin has been obtained; (*b*) in those cases of chronic affection of the eye in which a diagnosis is doubtful and in which therapeutic doses of tuberculin prove to be beneficial.

Tubercular affections of the eye may be roughly classified as follows:

Lids.—Lupus vulgaris and erythematosus, tuberculous thickening of the tarsus.

Lacrimal Apparatus.—Lupus of sac and canaliculi, conglomerate tubercle of the lacrimal gland.

Conjunctiva.—Lupus vulgaris; acute miliary tuberculosis following direct infection through a wound; thickening of tarsus and overlaying conjunctiva, more appreciable from the conjunctival surface. In tuberculosis of lids and conjunctiva the preauricular and cervical glands on the side affected are usually appreciably enlarged.

Sclera and Cornea.—Tuberculosis of one of these parts of the fibrous tunic of the eye is seldom present without participation of the other, except in cases in which the sclera posterior to the equator is involved. The disease is marked by chronicity; the inflammation is of a low form, the changes characterized by the development of more or less distinct foci, which in the cornea are pale, frequently having a yellow tinge in the sclera, varying according to the degree of thickening and hyperemia of the tissue involved. It seldom occurs that the entire anterior segment of the sclera is affected, or that the entire cornea is involved, and if this be the case, the degree of the process varies in different parts. An interstitial keratitis closely resembling that observed in syphilis may develop in the cornea, and a sclerosing effect may be produced. Some vascularity, usually superficially located, develops in the cornea. The affection of the anterior segment of the sclera is almost without exception an extension of a tuberculous process from the ciliary portion of the uveal tract.

Phlyctenular Keratitis.—Numerous recent studies of this disease appear to show a relation between the subacute or chronic forms of phlyctenular keratitis and conjunctivitis and tuberculosis. Tubercle bacilli have not been demonstrated in the lesions, but the occurrence of a positive systemic reaction to tuberculin in many of these cases, and the beneficial effect of tuberculin in some, suggest a strong interdependence between tuberculosis and these lesions.

Iris.—Tuberculosis of the iris manifests itself in four quite distinct forms. The most common is the develop-

ment of small, yellowish-gray nodules, scattered principally throughout the major zone of the iris. These occur in children and young adults as a result of active tuberculosis in some other part of the body. In a high percentage of the cases both eyes are affected. A second form is the conglomerate tubercle, which develops slowly and is accompanied by little inflammatory reaction, it is monocular almost without exception. A third is the tubercular granuloma occurring in children.

The three forms mentioned may be accompanied by the white, floating, "mutton-fat" exudation, which is observed, so far as I am aware, only in tubercular affections of the eye.

A fourth form is an affection of the iris which leads to heterochromia and partial atrophy, and is apparently due to ptomain absorption rather than to the actual presence of the bacilli in the iris.

Choroid.—This portion of the uveal tract may present the miliary or the conglomerate tubercle and the tuberculous granuloma. Also exudative and disseminate choroiditis of tubercular origin.

Retina and Optic Nerve.—The recurring hemorrhages in adolescents are apparently tuberculous in origin in many of the cases, as suggested by Axenfeld and Stock in 1909. Some forms of retinal exudation are tubercular in origin. A papilloretinitis with the development of conglomerate tubercles is also sometimes observed. This may be accompanied by quite extensive retinal detachment.

What kind of tuberculin can be used to best advantage in diagnosis and treatment?

Tuberculin "T. O." is a filtrate of tubercle bacilli culture grown on glycerin broth, concentrated by heat to one-tenth of its volume, and then filtered through porcelain to remove the germs. "T. O." is subjected to a temperature of 60° to 90° C., depending on the laboratory in which it is produced. It "contains the soluble products of the tubercle

bacillus in a 50 per cent. glycerin solution. It is thought that valuable immunizing properties are lost by the heating process to which it is subjected."

Tuberculin T. R. is made by triturating dried tubercle bacilli into a fine powder, treating the powder with a normal saline solution, and centrifuging. The suspension is thus separated into two layers, the upper layer containing the glycerin-soluble substances, the lower containing the substance left behind after extraction with glycerin. This latter constitutes T. R. Initial dose: $\frac{1}{5000}$ to $\frac{1}{1000}$ mg.

Tuberculin B. E. (bacillary emulsion) is a suspension of one part of thoroughly triturated tubercle bacilli in 100 parts of distilled water, to which equal parts of glycerin are added. Initial dose: $\frac{1}{5000}$ to $\frac{1}{1000}$ mg.

Tuberculin B. F. (bouillon filtrate) (Denys) is a preparation similar to T. O., except that it is not concentrated by high degrees of temperature. Initial dose: $\frac{1}{10000}$ to $\frac{1}{1000}$ mg.

Tuberculin T. B. K. (Béraneck) consists of the extracellular toxins of the bouillon culture, together with intracellular toxins extracted from the bodies of the bacilli with 1 per cent. phosphoric acid. Theoretically it should contain, as far as possible, all substances having immunizing properties, and avoid the danger, common to the emulsions, of containing live bacilli. "It is supplied in 15 different solutions, each one double the strength of the preceding one."

Tuberculins T. R. and B. E. are in a sense vaccines, and since they are not subjected to sufficient heat to kill living bacilli, may (although the trituration is supposed to be sufficiently thorough to destroy all living germs) contain living germs and subject the patient to the danger of infection.

It is generally conceded that tuberculin "T. O." is best suited for use for diagnostic purposes. Other tuberculins

can be employed, but the regulation of the dose, etc., has not been so definitely fixed and the results are not so uniform. Consequently their employment is not so satisfactory.

For therapeutic purposes T. R. and B. E. are more largely favored, and T. B. K. (Béraneck) is preferred by some.

Although theoretically the tuberculins prepared without heat are superior, since their immunizing qualities are not supposed to be impaired, practically I have obtained fully as good, if not better, results from the use of old tuberculin as supplied by the New York Board of Health as from any other of the tuberculins, and in some of the cases, at least, the T. O. tuberculin has been superior. This has proved to be the case so often that I have now come to employ the old tuberculin in almost all cases as long as I see improvement. If improvement ceases, I resort to the use of one of the other preparations—sometimes T. R., sometimes B. E. I have found the "B. E.," as put up in soluble tablets, to be fairly satisfactory, on account of the convenient form and its stability. In all cases when the tuberculin employed fails to improve the condition, it is discontinued and another preparation substituted.

In what dose and for how long a period should tuberculin be employed in treating tuberculosis of the eye? It has been my practice to employ tuberculin for therapeutic purposes in a dose just short of sufficient to produce a systemic reaction, and to repeat the dose every four or five days. A systemic reaction may be produced by a very small dose. Stillwell* reports a slight systemic reaction from an injection of T. R., $\frac{1}{4000000}$ mg., and a strong reaction from an injection of $\frac{1}{2500000}$ mg. The writer has employed T. O. in dose of 3 mg. in an undoubted case of tuberculosis without inducing any appreciable constitutional effect. The range of susceptibility or of sensitiveness to tuberculins is very considerable.

* Annals of Ophth., 1915, vol. xxiv, p. 413.

In regard to the length of time over which treatment should be continued, I think that it can be fixed only approximately. Some relapses will occur in spite of most careful management. It is the writer's custom to continue the injections for at least two months after all signs of activity of the tuberculous focus have subsided, and then to keep the patient under observation and to recommence treatment if any sign of relapse is discovered. Von Hippel* reports 243 cases of tuberculosis of the eye treated with tuberculin. Of these there were relapses in 32. The relapses were most frequent in tuberculosis of the uveal tract. Of 75 cases of tuberculosis of the iris and ciliary body, relapses occurred in 15.

In the writer's experience relapses in cases of tuberculosis affecting the uveal tract have been most frequent. Relapses in corneal tuberculosis were next in frequency.

DISCUSSION.

DR. SAMUEL D. RISLEY, Philadelphia: I should like to report one instance which seems to answer one of the inquiries in Dr. Weeks' excellent paper, *i. e.*, does old tuberculin react on other than tubercular growths? It dates back to one of my very earliest experiences in the treatment of tubercular affections, to a time when I was quite ignorant about the matter in many respects. It was then my habit to inject hypodermatically a sufficient dose of Mulford's old tuberculin to produce a general and local reaction if the patient were tuberculous. Indeed, the design of the initial dose was for diagnostic purposes quite as much as for therapeutic results.

During my vacation period my assistant surgeon admitted to the wards of the Wills Hospital a large, muscular, but not fat, Russian, apparently in excellent general health; he claimed, indeed, to have always been well. The right eyeball was proptosed—projected forward by an inflammatory orbital growth, which had been diagnosed as gumma of the orbit. There seems to have been no Wassermann reaction

* Graefe's Arch. of Ophth., vol. lix, p. 1.

taken, but he was placed on mercurial inunctions, and these were carried to the point of ptyalism, but without relief. On my return all treatment was stopped, his temperature taken four times daily for two weeks, disclosing a uniform rise of evening temperature; during this interval there came on an immense swelling, with tenderness and pain of his parotid gland on the same side, and, a few days later, a swelling of the ramus of the right jaw, intensely hard. He had no bad teeth or other means of accounting for it by existing local conditions. Observing the steady and daily rise of temperature, I gave him a diagnostic dose of one minim of old tuberculin hypodermatically. In twelve hours his temperature began to rise, and the following day went up to 103°, with marked aggravation of the local conditions. In a word, there was pronounced local and general reaction. His morning temperature went down in forty-eight hours, and he was relieved from pain in the orbit. The parotid swelling began to soften. I waited a week, and then repeated the dose, with almost similar results. When the reaction subsided, he was enthusiastic over the relief he had received. The proptosis of the eyeball was reduced, the hard growth in the ramus of the jaw was smaller and less tender, and then disappeared rapidly. The proptosis entirely disappeared after the third dose, and the man wanted to go out of the hospital and back to work again, which, when it was safe, he was permitted to do. I reported the case to the College of Physicians at the time. Two months later he returned to the hospital with a marked syphiloderm all over his body and arms. Then there was a very positive Wassermann reaction and the syphiloderm disappeared under mercurials.

Now this awakened in my mind a number of inquiries: Can old tuberculin produce a profound reaction in other than tubercular lesions? It seemed on study that this man was, notwithstanding the failure of mercury to relieve him, a syphilitic; and that probably the original diagnosis was right, the lesion being, in fact, a gumma of the orbit. While he got no relief from the antisyphilitic treatment, the profound reaction from the old tuberculin was followed by a rapid subsidence of the symptoms. The man was then placed on mercurial treatment. The general lesions disappeared, and he has been well since. He was kept under observation for a year or more.

DR. A. E. DAVIS, New York City: I wish to give my experience as to the size of the dose and as to the length of time of treatment in some of these cases. The size of the dose varied greatly with the patients: some took enormous doses with no reaction, while in others the smallest dose was enough to produce a reaction. I recall a case in a young girl about seventeen or eighteen years of age who had a keratitis and iritis, and reacted to the old tuberculin in all three phases —local, focal, and general. She reacted to both the von Pirquet and the subcutaneous test. It seems practicable to give the von Pirquet in both children and adults; and if this is not satisfactory to give the subcutaneous. We began therapeutic treatment with one minim doses of B. E., five-day intervals, increasing one minim a dose; after the fourth dose there was some reaction, but not much, to the therapeutic injections; the fifth dose (five minims of B. E., vial No. 1) caused the temperature to rise to 105° and the pulse to 100. There was marked swelling of the tonsils, the parotid glands were involved, and she had marked general reaction. They sent for me from the hospital, thinking that the patient had pneumonia; but, as she had pain in so many places, I decided against this view. After ten days the temperature returned to normal, and subsequently we could not give more than two minims of B. E. There was a satisfactory result, so far as the eye was concerned. The inflammatory process subsided in a few weeks, and she has been well for a year and a half.

I have had one other patient under constant treatment for five years—it was a case of choroidal tuberculosis in which one eye had been lost. The patient had been treated for syphilis for a number of years before coming under my observation. There was an active choroiditis. He was a man twenty-five years of age. He had a most pronounced reaction from the von Pirquet—local, focal, and general. He has been taking tuberculin for five years. In the fourth year he stopped treatment for two months and had a recurrence of the trouble. He was put back on the treatment with prompt relief, and is still on it. The interval of dose in this case is two weeks.

DR. HIRAM WOODS, Baltimore, Md.: What was the effect of stopping the treatment?

Dr. Davis: The patient gained 35 pounds on the treatment at first, stopping all other treatment. He maintained that weight until the treatment was stopped, then he began to lose flesh, to have flashes in the eye, and the vision to decrease.

Dr. Woods: You lose what you have gained when you stop?

Dr. Davis: Yes. As to the form of tuberculin, I am like Dr. Weeks in preferring old tuberculin for therapeutic use. I think that Dr. Verhoeff was the first in this country to emphasize the value of the old tuberculin as a therapeutic measure. I have given the B. E. mixture a great deal, but I think that we get better results from the use of the T. O. than from the B. E. mixture.

Dr. Hiram Woods, Baltimore, Md.: Some years ago Dr. Theobald Smith published* an article on Vaccine Therapy, and he answered quite a number of the questions which Dr. Weeks has brought up; for instance, as to the possibility of harm coming from tuberculin. He emphasized the fact that tuberculous troubles in general medicine have "hands off!" written all over them, so far as tuberculin is concerned, when there is fever or any sign of an active process. Similarly, I believe, in localized tuberculosis, to which ocular tuberculosis belongs, so long as there exists evidence of acute disturbance (for instance, active recent inflammations, descemetitis, circumcorneal injection, etc.), one is more apt to do harm than good with tuberculin. In mixed infection, where syphilis and tuberculosis are associated, the use of tuberculin insufficiently sterilized, and possibly containing live bacilli, may aggravate the associated condition. The place for tuberculin is in what Dr. Smith calls the "residual" of tuberculous infection. From our standpoint, eyes do not keep well: slight *recurrent* circumcorneal injection is observed; corneal infiltrates hang on or relapse from time to time, and there may be the scintillating light flashes mentioned by Dr. Davis (mild choroiditis), the patients complaining of "steam" coming from the ground. There is some chronic disturbance going on, which does not get well and will last indefinitely—they are the ones, according to Dr.

* Jour. Amer. Med. Assoc., lxx, 1591.

Theobald Smith, as I interpret him, in which tuberculin helps.

Now as to harm: I reported two or three cases before the Academy of Ophthalmology and Otolaryngology in 1916 in which I believe harm was done. The first patient was a woman—one of my earliest cases. She got too much tuberculin, I know now, although the diagnostic dose was smaller than some are giving today. She had old central choroidal atrophy in one eye, and in the other an acute choroidal process. Having excluded syphilis and everything else, I gave 1.5 mg. of old tuberculin. There were within twenty-four hours elevation of temperature and pain and, worse still, a definite reddening of the foveal area in the better eye (the one acutely inflamed), near the point of an old healed exudate. Nothing occurred in the lost eye. Her vision was reduced from 20/40 to 20/200, and never got better. Did the tuberculin do it? If so, how? I ask that because, some time later, a man thought that he had found a positive Wassermann. I do not know whether the tuberculin caused excessive reaction near a healed tubercular spot or aggravated something else—stirred up a quiescent syphilis.

I saw a girl with a choroiditis in the nasal periphery. She had a large exudate. After the injection of $\frac{1}{1000}$ mg. of old tuberculin a fresh process started near the other, but it did not get up to the fovea. That healed and she got well.

I have a patient who, for six years, has been taking moderate doses of tuberculin,—very small doses,—but when the tuberculin is stopped, her "flashes" begin. She becomes nervous and fidgety, and is almost like a person without dope who is dependent on it for comfort. Also, after she has been two months without tuberculin, she will develop those symptoms that we see with a mild choroiditis.

DR. W. GORDON M. BYERS, Montreal, Can.: I should like to say at the outset that it is my conviction that tuberculin treatment is a two-edged sword, and that it should not be given by men who are unfamiliar with the subject; nor should it be administered until the whole system has been carefully investigated by an internist or competent practitioner.

My experience agrees entirely with that of Dr. Weeks, inasmuch as I have never seen harm follow a tuberculin

reaction. On the contrary, I have always noted that it did good. There are, however, many instances on record where a tuberculin reaction has done harm; and one should proceed, for this reason, very cautiously.

Chiefly as the result of an experience of my colleague, Dr. Tooke, we now consider that one milligram of T. O. as an initial dose is too high. A quarter milligram dose, in a woman of twenty-six years, whose blood and spinal fluid had been tested for syphilis with negative results, gave a decided local and focal reaction.

I think that Dr. Weeks would lay it down with me as a principle that, if a supposedly tubercular eye does not act favorably to tuberculin treatment, a reconsideration of the diagnosis is necessary; for it has been my experience that, in ocular tuberculosis, tuberculin properly given seldom if ever fails to do good.

Dr. Woods has brought up the point of the danger of treating these patients with tuberculin, especially in acute cases. My confrères at Saranac Lake, with whom I am constantly in touch, and my brother, Dr. J. R. Byers, in charge of the Laurentian Sanitarium, Ste. Agathe, P. Q., lay great emphasis on the importance of allowing cases to come into a more or less chronic condition before tuberculin treatment is instituted, taking advantage of the interval to build up the natural combative forces of the body by the usual hygienic measures, such as good feeding, rest, fresh air, and the like.

My preference is always for the bacillary emulsion; and we lay a great deal of stress on having it freshly prepared, and, of course, on methods of sterilization.

DR. EDWARD JACKSON, Denver, Col.: There are two or three points to which I should like to allude. One is the choice of the tuberculin. I have a distinct preference for the old tuberculin for therapeutic as well as for diagnostic uses.

Another point that I believe is extremely important, and not generally recognized, is that enough time should elapse between doses. It seems to me not only that the tuberculin should not be used in the relatively acute cases, but that the changes that are produced by one dose of tuberculin should pass from the acute to the chronic condition before

the dose is repeated. My rule is not to give the dose oftener than once in two weeks to start with; and not more than once a week in any case; and some cases have done better with three or four weeks between the doses than with the two weeks' interval.

With reference to the time of treatment: I think that these cases must be watched, and often treated from time to time as long as they have tuberculosis; and with some, that will be as long as they live. I have now a patient that has been treated for seven years in all, taking tuberculin for a considerable part of that time. Within the last year she has had a recurrence of iritis.

I have another case that has now been treated for five years. The patient came to Colorado fifteen months ago with a recurrence.

I want also to emphasize the importance of the point that Dr. Byers alluded to—the general treatment of the case along the line of tuberculosis; and that it is impossible to get good results without attending to that.

One other point is that the dose can produce permanent harm. In one case of intra-ocular hemorrhage in connection with disease of the retinal vessels I saw, on three separate occasions, fresh hemorrhages following within forty-eight hours of the dose of tuberculin; and in other cases I am sure that there has been some aggravation of the symptoms after what were, for them, excessive doses, although comparatively small doses as ordinarily estimated.

DR. S. LEWIS ZIEGLER, Philadelphia: Some twelve years ago I presented a case before this Society of tuberculosis of the cornea which I had successfully treated by repeated doses of old tuberculin. I agree with the view that the old tuberculin is the best tuberculin to use. As to the length of time and the dosage, I think that small dosage is certainly of the greatest advantage. The interval, as a rule, has been one week. The lesions that I have seen, and I have treated upward of fifty cases, have been mostly in the cornea and sclera. I recall, however, one marked case of involvement of the choroid that passed on to cataract. The course of the lesion could be watched, and both cataracts were later extracted. The patient never had satisfactory vision, owing to the destructiveness of the choroidal lesion, but it proved to be useful.

In most of the cases of use of tuberculin there was a marked early reaction; but the reaction was soon dissipated. The cases passed on, after that, without reaction, but the injections were continued for a long period of time, so as to be positive that the lesion had been eradicated.

In reference to the point made by Dr. Byers that there should be coöperation with the general practitioner in the treatment of these cases, I believe it is difficult to follow such a plan, for the reason that the majority of internists are very skeptical in regard to the use of tuberculin for this purpose.

DR. BYERS: Not for local lesions.

DR. ZIEGLER: Well, they expressed their opinion to me in that way, and, of course, one feels that their antagonism is the same for local as well as for general tuberculosis.

Dr. Weeks referred to the influence on the lymphatic system. I think that in all these injections, whether of diphtheritic or other serum, it is more or less the antibodies that bring about the result, and where there is sluggish lymphatic action, the use of tuberculin or of some other serum is followed by a distinct lymphagogue action.

DR. F. H. VERHOEFF, Boston, Mass. Eleven years ago I was a strong advocate of tuberculin treatment for chronic ocular tuberculosis, but I have changed my views entirely since then. My belief in the value of this treatment was due to insufficient clinical experience with the disease. I thought then that when we had a case of chronic ocular tuberculosis it would do badly if let alone. But the fact is that the majority of cases do well, and it is only the exceptional case that does badly. I find that in the very bad cases, in which you most want tuberculin to do good, it utterly fails, or perhaps makes them worse. I abandoned the use of tuberculin, and found that the cases did well under simple hygienic measures. The most important thing is rest. Locally, I give only atropin; often Fowler's solution as a tonic. I think that tuberculin, even for diagnostic purposes, is seldom necessary now that we know the clinical picture of ocular tuberculosis. Occasionally you get a case in which you are not sure of the diagnosis, and you can then use it.

I suppose that damage is done occasionally by diagnostic injections, although personally I have not had any serious

results. I have seen a metastasis occur in the cornea, a short distance from a focal lesion, evidently due to the tuberculin reaction; and I should suppose that a similar metastasis could occur in the fundus also, where it would do more harm. I have found that you can get a systemic reaction and a focal reaction in the eye in cases of ocular tuberculosis by injecting Coley's toxins. Probably injections of milk and other foreign proteins would do the same. I do not say that tuberculin may not hasten recovery in some of the very sluggish cases of ocular tuberculosis by producing focal reactions, but it is like splitting hairs to determine this point. Personally, I have become convinced that there is not sufficient to be gained in running any risk by using tuberculin, so I have entirely abandoned it for therapeutic purposes.

DR. JOHN E. WEEKS, New York City: I simply want to say, in regard to the treatment of tubercular affections of the eye, that the cases in which I find the use of tuberculin necessary are progressive cases. The theory, of course, in regard to the bad effect of tuberculin in acute general tuberculosis is that the system is so dominated by the toxins of tuberculosis that it has not the power to produce antibodies to combat these toxins; and that it must be allowed to recover that ability, if it will, in the presence of the tuberculous process, in order to make tuberculin of any value. When we have tuberculosis of the eye, there is, as a rule, a localized tuberculous process. Under such conditions the system is not so saturated with toxins that antibodies cannot be formed; and the accepted theory is that the antibodies are formed and that these toxins are combated. I fail to see any reason for saying that a tuberculous process must be inactive in order that it may be influenced by tuberculin.

The statement that the last speaker has made with regard to the use of tuberculin does not seem to be of very great value. I have had three cases referred to me for enucleation, advised by the people who sent them to me—these patients have at present very good eyes because the careful use of tuberculin was persisted in.

SOME OCULAR CONDITIONS AFFECTING THE EFFICIENCY OF THE AVIATOR.

COLONEL WILLIAM H. WILMER, M.C.N.A.

The medical problems involving the aviator are unique. The airman is compelled to spend much time in an unaccustomed environment. In addition to the stress of conflict common to all branches of military service he is compelled to subject himself to sudden changes—so sudden that there is but little opportunity for adaptive adjustments to occur. There are sudden changes of temperature, barometric pressure, muscular and mental exertion, from ground to labile equilibrium, from normal sea-level oxygen tension to a tension much decreased.

Under conditions of lowered oxygen, certain well-known physiologic changes occur which are adaptive in nature. There is a cry for help from the tissues of the body, which demand more oxygen. The blood-stream is not only a sewer carrying off waste products, but a nutrient fluid and a carrier of oxygen. This appeal for succor is met in good subjects by an increase in depth of respiration, by the dilatation of peripheral vessels, and by more rapid heart-beats. More frequent respiration and an increased blood-pressure are clumsy attempts at adaptation, and the airman soon breaks under such tension. In the blood itself there are changes consisting of concentration and an actual increase of available red blood-corpuscles.

At the Medical Research Laboratory the conditions of lowered oxygen tension are produced artificially by two different forms of apparatus:

(1) A large low-pressure chamber from which the air is removed by an electrically driven pump, the oxygen tension and the barometric pressure being simultaneously lowered, while the amount of CO_2 increase in the chamber is negligible, owing to the good ventilation.

(2) The Henderson rebreathing apparatus, where the subject breathes up his own oxygen from a tank, the excess of CO_2 being absorbed by sodium hydroxid.

Tests made by these different forms of apparatus correlate so closely that we are compelled to believe that the physiologic changes noted are due to the want of oxygen and not to the lessened atmospheric pressure. This is further proved by the fact that the administration of oxygen causes a quick return of the disturbed functions to the normal.

It is difficult to say how much of the disturbance of function is due to suboxidization in the central nervous system and how much is due to changes in the muscles themselves. At all events, the results are strikingly similar to the toxic effect of alcohol, etc.

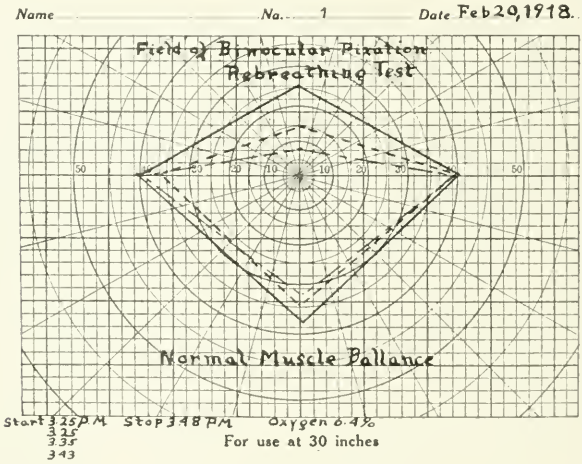
In various accident reports from aviation sources there is constant reference to the statement by pilots that vision was blurred during flight. In many cases the instruments could not be read correctly, distance from the ground properly judged, etc. We have, therefore, been interested in making our tests under conditions of lowered oxygen pressure to see just what changes do occur in the various functions of the eye. It is well known that the visual requirements for the air service of the United States are much more strict than those of any other nation, and there have not been many instances where careless selective examinations have passed candidates with defective vision or muscular imbalance. The fact that the ocular functions (especially the motor apparatus) do fall off in an atmosphere of reduced oxygen pressure convinces us of the continued necessity of high entrance requirements. The acuteness of vision, of course, is

the most important ocular asset of the aviator. In testing this function under low oxygen conditions, a number of factors have to be considered, such as practice, suggestion, concentration, etc. On the whole, we have not found any *very marked* change in the acuteness of visual perception. The great variations have been due to the falling off in the power of muscular adjustment. For instance, in hyperopic individuals there is likely to be a deterioration owing to the reduction in the power of the ciliary muscle. On the other hand, in those slightly myopic there is a tendency toward improvement for the same reason. Our experimental and practical experiences have not borne out the statement of some of our confrères that vision in the air is improved at the height of about 200 meters, owing to a congestion of the head, the choroid, and the retina. To test this we tried the effect of nitrite of amyl on the sight of a number of observers. There was falling off in vision in every instance except in one myopic subject, where there was improvement.

Normal color vision is most important for the aviator; but we have found very little practical change in this function, though there have been found some alterations of its threshold values.

A large field of vision is very necessary for a good aviator. This is especially important because the fuselage causes such a large blind angle in the visual field. In the tests of the visual fields, some rather definite changes have been noted, in some instances indicating that the periphery of the retina is much more sensitive to oxygen want than the macular region, either through the nervous apparatus of the retina itself or through suboxidization in the perceptive centers. While in the average we have not found any great change, in some cases up to 10,000 feet the visual fields seem to increase; about 15,000 feet there is a slight contraction; at 20,000 feet there is a marked contraction for form and color.

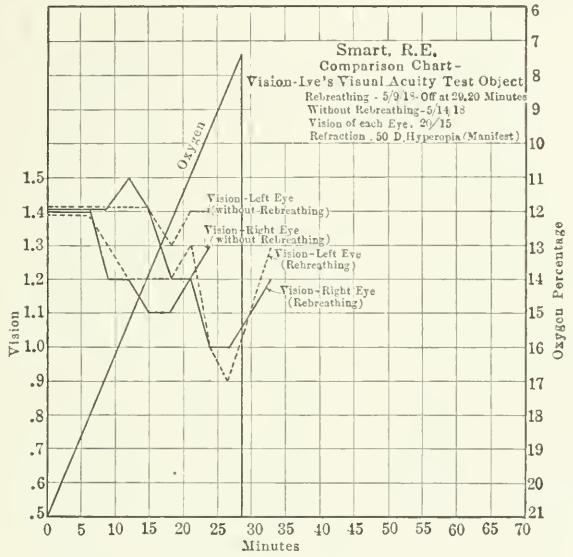
Retinal sensitivity is not tested in the original selection of the aviator; but later, in his classification, this test is essential, and it is also necessary to determine whether this function is diminished under certain air conditions. The test for threshold retinal sensitivity is interesting, and, for laboratory purposes, a very practical and exact test. However, it requires too much time to be useful as a quick clinical test. So, in substitution for this, we use a comparative sensitivity test. In this test the object is a gray letter on a



lighter gray background, there being 13 shades of perceptible difference between them. One eye is closed and the photometric wedge is passed before the other eye until the letter blends into the background and can no longer be recognized. The point at which this occurs is noted on the millimeter scale of the wedge. The time allowed for the process is from five to eight seconds. It is repeated three times for each eye. The wedge that is used was devised by Lieutenant Reeves. It consists of a layer of gelatin of increasing density of color between two pieces of optical glass. The light transmission

has been calculated for each millimeter on the scale, and the normal has been established by repeated tests at the laboratory.

The function of perception of light and motion is among the first developed of all ocular percepts. The quick and accurate perception of motion is very valuable to an aviator. As has been stated before, Nungesser, the famous French

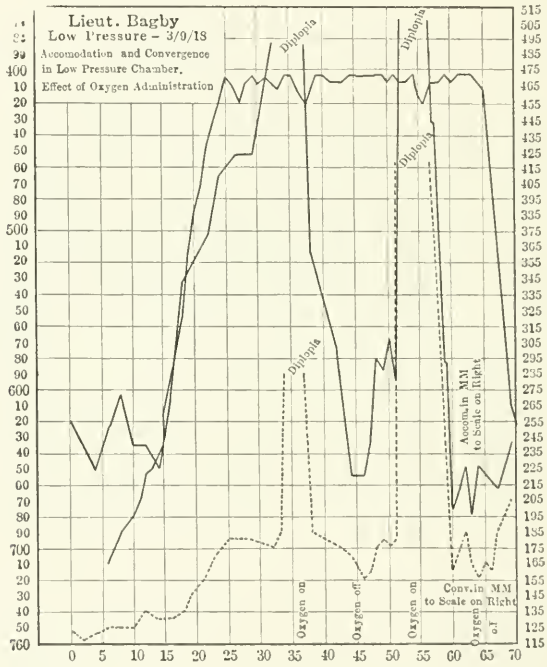


ace, said: "You need eyes all around your head, and after a time you get them."

With the subject placed 15 inches from the test screen, the threshold perception of motion covers a field about five degrees larger than that for the detection of form, but our tests at the laboratory show that the *field for the accurate* perception of motion direction is usually about three degrees less in all quadrants than the field for form, as *usually* taken.

Stereoscopic vision does not, according to our experience, suffer any very serious impairment in oxygen deprivation.

The test for the simple visual reaction time has not appeared to us to be of much practical value. In this test the one possible reaction to the one possible stimulus becomes practically an automatic reflex. However, the Reeves visual discrimination reaction time test is of much ophthalmopsychologic value. In this test there are four possible correct reactions to five possible stimuli. This act involves the

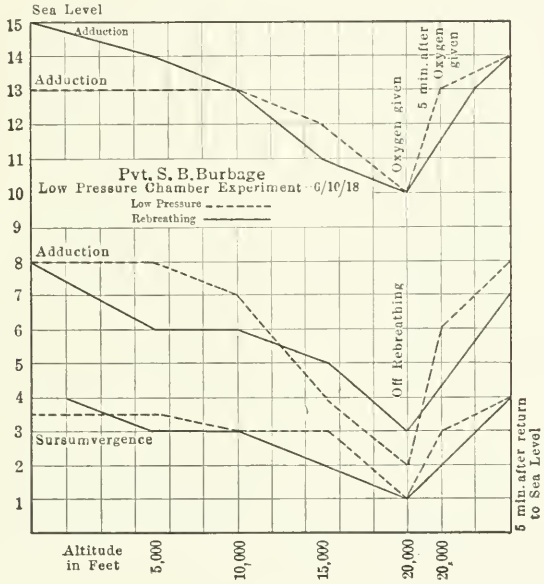


higher mental processes and cannot become a simple reflex. In the laboratory the average reaction time of this test is one-half second, while the time given by many observers for the simple visual reaction is one-nineteenth of a second.

After all, the serious ocular disturbances produced in the peculiar environment of the airman take place in the intrinsic and extrinsic muscular apparatus. There is in many cases a

falling off in the power of accommodation, convergence, and, naturally, in the field of binocular fixation. To illustrate this point, several diagrams are appended.

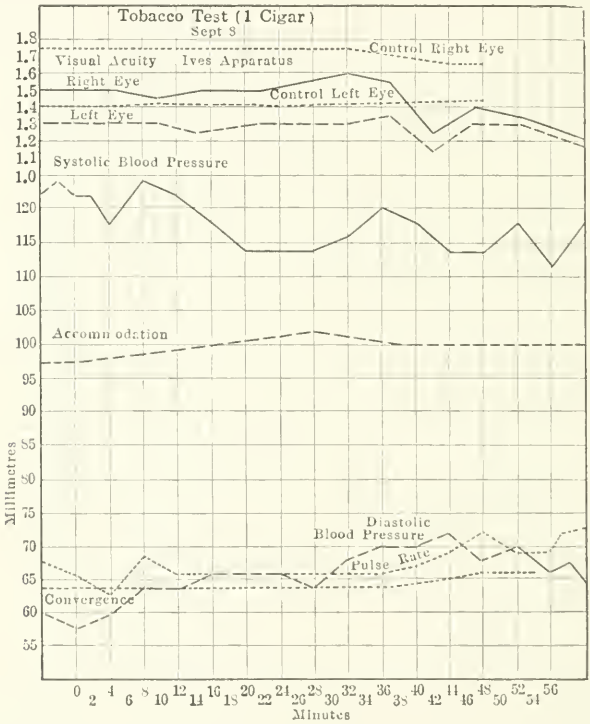
With the failure of accommodation it is impossible to read the figures upon the ever-increasing number of instruments in the fuselage. The pilot has to shift his gaze quickly from the great expanse around him to the cockpit. Therefore, his accommodation must be not only accurate but free from ab-



normal lag, otherwise the penalty is a broken machine, with the injury or death of the pilot. So, too, the failure in convergence means double, confused, and a danger-breeding vision.

While it does not bear directly upon the problems of aviation, as a bi-problem, the intraocular tension has been tested under conditions of much reduced barometric and oxygen tension. In these tests no correlation has been found between

these conditions, nor between the intraocular tension and the varying blood-pressures occurring in a rarefied atmosphere. But more closely related to the problems of aviation are the effects of nicotin (or allied substances) upon the ocular functions. With reference to these effects upon the eyes and blood-pressures of non-smokers, 16 subjects have been tested

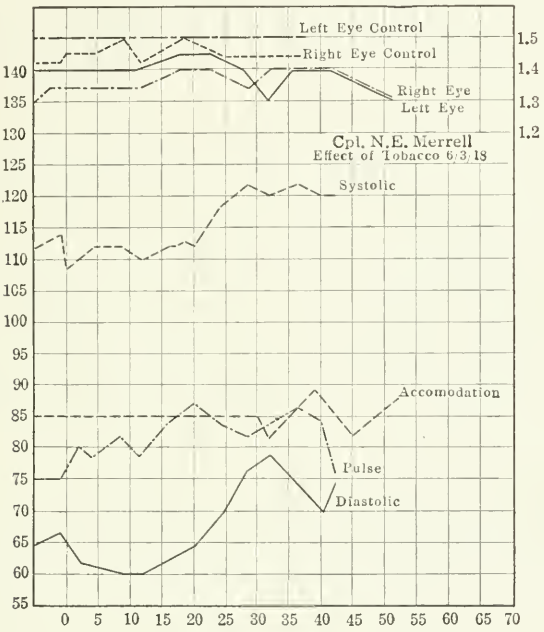


in the laboratory, the smoking of one cigar being the time limit of the test. In 12 cases there was loss in visual acuity; in one there was gain; while in three there was no change. In about one-half of the cases there was a falling off in the motor apparatus of the eye. In regard to the interesting subject of blood-pressure, it is sufficient to say, without going

into details, that about 69 per cent. of the cases showed a rise in both systolic and diastolic blood-pressure.

After months of serious thought and work on aviation problems, we have asked ourselves:

After all, what conclusions can one draw from this experience with an organ so wonderful in its fine adjustment that it can be compared, as Reeves has said, to a marvelous



balance that can, "with equal accuracy, weigh a hair or a ton of coal"?

While the eye is only one of the peripheral sense organs concerned in the function of equilibrium, it is second to none in importance. We have found that its proper functioning is affected by oxygen want apart from the other trying conditions of flying, and we are more and more impressed with the vast importance of the initial selection of candidates who

possess good eyes and well-balanced muscles, who have good adduction, and who are practically free from hyperphoria. Equally important is the proper classification of the cadet, that he may be assigned to the type of work for which these laboratory tests have shown that he is best fitted. A cadet with low retinal sensitivity could not be a night bomber. A candidate who fails to read his instruments correctly and sees double at 10,000 feet could not be a pursuit or combat pilot. Once classified, the pilot should not be lost sight of. The unceasing care of the flyer is the culminating duty of the flight surgeon.

A slack wire on a control is not so dangerous as a poor convergence. After the strain of service a tendency to a weak muscle becomes a manifest error. A weak convergence becomes a dynamic divergent squint.

By the aid of laboratory tests and by the constant, watchful care of the flight surgeon and of the physical director, many cases of incipient staleness can be detected and prevented, much economic waste can be prevented for the Government, while valuable young lives will be saved.

DISCUSSION.

DR. ALEXANDER DUANE, New York City: I think that what Dr. Wilmer has touched on is applicable to other classes of service besides aviation; for instance, signal work, in which I am particularly interested. It concerns men in this service, not to the same extent, perhaps, but at least in similar ways. Dr. Wilmer spoke of the great necessity of color vision in aviators. I should like to ask whether it is color vision *per se* that is required, *i. e.*, the ability to distinguish colors as such, or rather the light sense that is in question. I was asked to examine a man who was coming up for the naval aviation service, and he was very definitely color blind; and, being such a good man, they asked me to have another look at him and see whether he could be passed. I had to report that he was decidedly color blind to every test; but that, nevertheless, I thought that he could be tried

practically to see whether he would possibly do. I did so for two reasons: First, that he had already had a year's flying, and a very successful one; and second, that he was able to make extremely fine distinctions of light and shade, and apparently of color. Of course he could not tell the colors as we do, but he could make distinctions at considerable distances. He told me that he never had the slightest difficulty, when coming down, in recognizing a patch of green field, a patch of plowed field, etc. It occurred to me that our tests of the color sense might not be so useful as we think. We might, because of them, reject a man who would be valuable.

DR. WILLIAM H. WILMER, Washington, D. C.: A certain percentage of color-blind subjects do slip past the examiners and get into service. For instance, at the laboratory we have just discovered a pilot who is red and green blind. He passed the examining board by previously learning the Jennings color test. He is very useful as an instructor, but he could not make maps or be a combat pilot. Good color vision is essential to recognize the markings of various planes, the colors of roofs, chimney-pots, lights at airdromes, etc. Of course, there is not necessarily any correlation between color vision and the luminosity sense. The latter is frequently acute in subjects who are very weak in the color sense. The enemy cleverly flies early in the morning, forcing our men to fly with the sun directly in their eyes. The pilot must wear tinted lenses, but it is important that the glasses should not seriously change the color values. With these lenses the pilot should be able to recognize even the differences in the color of the various uniforms, etc.

THE INERTIA OF ADJUSTMENT OF THE EYE FOR CLEAR SEEING AT DIFFERENT DISTANCES.

C. E. FERREE, PH.D., AND GERTRUDE RAND, PH.D.,
Bryn Mawr, Pa.
(By invitation)

By inertia of adjustment is meant here the lag in the action of the extrinsic and intrinsic muscles in adjusting for clear seeing at different distances. The amount of lag in this function is found to vary a great deal from individual to individual. In the results to be presented, the minimum time required to change from the adjustment for clear seeing at or near the near-point to that for clear seeing at six meters, and the converse, has been measured in several cases. So far the investigation has been conducted primarily as a study of the method, with special reference to its applicability as a test for fitness for vocations for which speed and accuracy of adjustment are a prerequisite. In this particular especially the writers believe that the aviator must excel. The rapid development of the science and art of aviation, brought about by the present war, emphasizes the need for tests which will facilitate the selection of the supernormal eye. It is scarcely to be expected that the conventional clinic tests, designed more particularly for the separation of the subnormal from the normal eye, are fully adequate for this purpose.

It will be obvious, without discussion perhaps, that in estimating the fitness of an instrument, apparatus, or human organ for a particular task or for the range of work which it may be called upon to perform, other aspects besides maximum power or capacity of response should be taken into account. Some of these are lag, steadiness or stability of

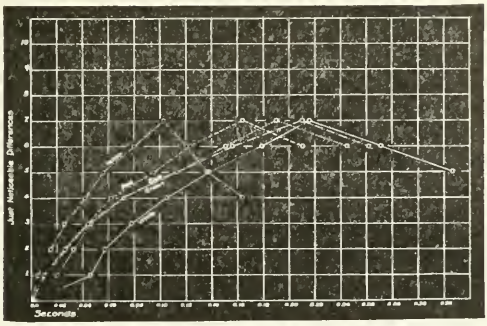
response, power to sustain response, rate of fatigue or delay of response, rate of recovery, etc. All of these functional aspects are present in particular in case of the eye, and their variation from time to time in a given eye and from eye to eye can be measured with a degree of precision that is adequate at least for many comparative purposes. As has already been indicated, we have been concerned in the present work with only one of these aspects, namely, the lag in the eye's reactions to its stimulus. In the study of this phenomenon at different times in our laboratory, three subdivisions have been made: the lag in the response of the retina to colored and colorless light and its change with change in the intensity of light, the lag in the adjustment of the eye for clear seeing at different distances, and the lag in the development of the perception of depth or distance. The relation of all of these to the functional efficiency of the eye will be considered very briefly in passing.

Because of its small order of magnitude, the first of these types of lag is of comparatively little importance in most of the uses to which the eye is put. In all acts of seeing, for example, in which a change of adjustment is required, the lag in the retina's action is insignificant in amount as compared with the lag in the muscular action. It becomes of importance only in such uses of the eye as permit of a very short exposure to its stimulus, usually with the muscular adjustment already made, or in cases in which it is important to have the maximum response of which the eye is capable. Examples of the former may be found in various uses of the eye in the technical work of the laboratory; and of the latter in signaling with colored and colorless lights. In order to show something of the order of magnitude of the lag in the retina's response and its variation with the wave-length and intensity of light used, we have constructed curves in which the sensation as it rises to its maximum value is plotted in just noticeably different steps against time of exposure.

These results were obtained in our laboratory three years ago in a comparative study of methods of determining lag.* Four of the most promising of the older methods and three new ones were used in making the determinations, and the results of these methods were checked against each other. The comparative study was made throughout on the same observers. The dominant motive in making this study was to find or devise a method which would have sureness of principle and precision and at the same time the feasibility that is needed for practical applications. One of the new methods was found to answer surprisingly well to these requirements, considering the nature and difficulty of the problem. The lights employed were narrow bands in the red, 686$\mu\mu$; yellow, 588$\mu\mu$; green, 511$\mu\mu$; blue, 463$\mu\mu$; and white light. The colored lights were taken from the prismatic spectrum of a Nernst filament, and the white light was synthesized from this spectrum. The lights were all made photometrically equal; and, in addition, for the sake of more absolute specification, their physical intensities were measured by means of a thermopile.

The results obtained showed that observers differ in all the following regards: amount of lag for a given stimulus, the amount of difference in the lag for the different wavelengths and for white light, and the effect on these differences of changing the intensity of light. In the case of the observer whose results are given in Fig. 1, three intensities of light were used—0.057, 0.151, and 1.21 meter-candles. It will be noted that for the lowest of these intensities the rate of rise to the maximum was in the following order: yellow, red, blue, green, and white, the time of the maximum ranging between 0.1 and 0.22 second. For the highest intensity the rates of rise were in this order: green, yellow, white, and red; the time required to reach the maximum ranging be-

* We are indebted for these results to M. A. Bills, a former graduate student.



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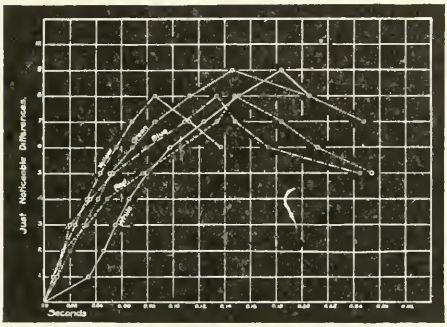

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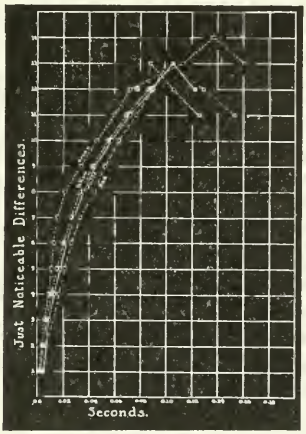


C

Fig. 1.—Showing the results of a determination of the lag in visual sensation in its relation to wave-length and intensity of light. In A the lights employed were made photometrically equal at 0.057 meter-candle; in B, at 0.151 meter-candle; and in C, at 1.21 meter-candles.

tween 0.085 and 0.14 second. Blue was omitted from this series because it could not be obtained at the required photometric value. At the intermediate intensity, a transition condition is shown which can best be gotten from the charts. That is, in passing over a certain range low in the intensity scale to points higher in the scale, there is a radical change in the order of rate of rise tending toward a complete reversal at high intensities; while at intermediate points in the scale the lag is shown to be in a state of transition between what is present at low and at high intensities. An increase in intensity is found also to lessen the lag very considerably or to increase the rate of rise. These details, however, are much more important in certain phases of the use of the eye in laboratory technic, for example, than they are in immediate relation to the work of the present paper. Our purpose in introducing them here is, as we have already indicated, merely to give some general idea of this feature in the eye's slowness in responding to its task, and in particular to show that, as compared with the inertia of adjusting clearly to receive its impression, the inertia in its sensory reaction is relatively unimportant for most of the work which it is called upon to do.

So far our investigation of the lag in the perception of depth has been made in stereoscopic vision, and for an entirely different purpose than the grading of individual capacities. The results show, however, that depth comes into the percept later than height and breadth and color and brightness, and that the amount of lag varies for different observers. Whether this type of lag could be made a feasible basis for the grading of individuals for vocational purposes we are not prepared at this time to say. Speed and accuracy in judging distance are doubtless important items in the qualification of an aviator, for example, and it may be possible to work out feasible tests for certain fundamental aspects of the ocular foundation of this ability. Indeed, the lag in the

adjustment of the eye for clear seeing at different distances should sustain a somewhat fundamental relation to speed of judging distance, since both the adjustment of the eye and the clear seeing of objects are in general the important ocular functions involved in the judgment of distance. Unfortunately for our purpose, however, the judgment itself is not an ocular function. The eye provides only the criteria,—and a very complex set of criteria at that,—from which the individual learns by experience to form his judgment of distance. The testing of these extra-ocular capacities, the ability to estimate distance, more especially under an entirely new set of conditions for which definite standards or patterns are wanting, is perhaps just as important as the testing of the ocular capacity itself. The testing of the ocular capacity, as registered in certain simple space judgments, with or without the element of time or speed of performance, is capable of definite treatment. All that can be said of the remainder of the problem is that it is open to investigation.

On quite a different methodologic plane, however, is the determination of the lag in the adjustment of the eye for clear seeing at different distances. The making of these determinations by the method we have used involves no extra-ocular capacities of a higher order than are required in the acuity tests for illiterates. Moreover, a direct objective check is applied to the subjective judgment. That is, the letter E, built to scale and turned in different directions, is used as a test-object for the different distances, and the observer is required only to indicate the direction in which the letter points in any given case. Such testing of human functions, even without the objective check, is, so far as method and principles of testing are concerned, just as definite as the testing of those physical instruments, the responses of which must be read by the eye from a moving pointer and scale or their equivalent.

Our purpose in making these tests has been, as we have

already indicated, primarily to ascertain whether eyes rated by the clinic tests as normal or approximately so cannot be more finely graded as to their working efficiency or fitness for special purposes, when other important functions than those considered in the clinic tests are taken into account. For this purpose we have aimed, therefore, to test, for the greater part, only eyes that have been passed in the clinic as normal, or as having defects so insignificant as not to need correction. Ninety-four per cent. of the eyes of this group were able to read quite readily at six meters under 5.2 foot-candles of light, the test-type designed to be read at four meters; and the remaining 6 per cent., the type designed to be read at 5 meters. In addition a few were tested whose eyes were corrected by glasses. This was done for the purpose of getting results comparative of the performance of eyes corrected to standard according to the norms of the clinic, and the uncorrected normal eye. All but one of the uncorrected group were between eighteen and twenty-eight years of age, and only one of the corrected group was over twenty-eight. Three of the observers had worked pretty steadily for a year or more with high-power microscopes, four were trained in the observations of physiologic optics, and the remainder were selected at random from the college community. The best results were obtained from one of the three who had been trained in the use of a high-power microscope, but her results were closely rivaled by those of a college sophomore whose eyes and observational powers had received no special training. However, the results of the three whose eyes had received special training in the use of the high-power microscope averaged rather strikingly high. To what extent speed of adjustment can be trained is yet to be determined.

Fortunately, for the feasibility of the test, the immediate practice effect is low; or, more properly speaking, it is rather high in the initial observations, but soon ceases to be troublesome. Also the precision for any given set of determinations

is high. As might be expected, though, there is a diurnal variation in the results corresponding to the diurnal changes in the function tested. The maximum range of these variations, however, is small as compared with the range of variation between individuals. It is not great enough, so far as we have thus far been able to determine, seriously to affect the grading of eyes with sufficient precision for practical purposes on the basis of the tests taken at any one time chosen at random. The fact that there is a diurnal variation suggests, however, that if the test be used as a check on fitness for aviation, it might be of advantage to determine each individual's norm and require a short test before each flight, or as often as may be needed to keep track of the variations and to safeguard against the more serious depressions that may occur.*

The working distance for the far object was chosen as six meters and for the near object as 18 cm. The far object at this distance subtended a visual angle of 7 minutes; the near object, 14.8 minutes. In choosing both the working distance and the visual angle for the near objects, care was taken not to approach too closely to the limiting values. This was

* If a check is wanted on the diurnal variations in other motor coördinations, as well as those of the eye, a reaction experiment involving choice may readily be combined with the ocular experiment. Two forms of this reaction experiment are suggested: (1) A four-finger reaction key may be used, each of the keys to indicate one of the four possible positions of the letters—up, down, right, and left. That is, the observer, just as soon as each letter—near, far, and near—is discriminated, indicates the direction in which it points by pressing the proper key. (2) A wider range of coördinative ability could be tested by having two keys operated respectively by the right and left hand, and contacts by the right and left foot. By soldering contacts on each edge of the exposure discs in circuit with an electromagnetic marker, and by having the reaction keys and foot contacts also in circuit with electromagnetic markers, the length of exposure for each test object and the reaction times could all be recorded simultaneously on a kymograph, together with a time line traced by an electric tuning-fork. With these records as a check on the quickness of the motor functions of eye, arms, and legs, and the mental functions involved in choosing, it would seem scarcely possible that the aviator could grow stale or suffer even temporary depressions of any consequence without the knowledge of the laboratory corps. Also from the accumulated records a comparative rating of the stability of the men could be made. It may be, of course, that the eye records alone will serve as a reliable index of the variations in the observer's general condition, but that correlation has not as yet been investigated.

needed to safeguard the results against individual differences in the near-point and in acuity. That is, it was found that unless these limiting values were too closely approximated, small variations either in the visual angle or in the working distance produced little difference in the results.

Since our problem was in part to devise and try out apparatus, the determinations have been made with two types of apparatus, one of which is slightly simpler in construction and use than the other. By means of the more complicated apparatus, however, a more complete analysis of the problem is possible. That is, by means of the simpler apparatus it is possible to make the following determinations: the lag of perception with the eye in approximate adjustment for the near object, the time required to change from this adjustment to that required for far seeing, and the time required for the double excursion, *i. e.*, from near to far and back again to near; while by means of the other apparatus we were in addition able to break up the double excursion into its two halves, the time required to pass from near to far and back again to near. By means of the second apparatus, moreover, all the determinations may be made in a single swing of the compound pendulum governing the time of exposure.

There is another advantage of the second apparatus which came out quite plainly whenever a comparison of results was possible on the same observer. That is, in its use a provision was made to cut off the test-object just as soon as it was discriminated. The eye was not allowed to linger on the far object, as was its tendency when the double excursion was not broken up into its two halves. The value of the double excursion, as determined directly by the first apparatus, was, for example, appreciably longer than when it was determined by adding together the values of the two halves as determined by the second apparatus. This tendency of the eye to linger where it can, thus not only makes a difference in the absolute value of the results, but there is a

danger that it may affect also the comparative values. That is, the latitude offered not only gives a chance for a variable performance or a variable error from time to time with the same observer, but it leaves the results open to the influence of this factor in case of different observers.

Obviously, the test may be used in two ways: (1) Records may be made of the maximum performance of each individual. This would be the analogue of making acuity tests in terms of the minimum visual angle each observer is able to discriminate. This procedure is the longer of the two, but results in a much finer grading of performance. (2) Two, three, four, or any suitable number of levels of performance may be chosen, and the apparatus set at once to give these levels. This method of testing would roughly place individuals into ranks or groups and is the analogue of the Snellen method of grading acuity. It is a much quicker procedure, but the grading made is correspondingly rough. By this method the ranking or testing of a given individual by a practised person should occupy but a few minutes. The results given in this paper were obtained entirely by the former method. It is obvious that the latter method could not be used until sufficient work had been done by the former to establish the required norms for the vocation or purpose in question. Results obtained with the two types of apparatus are shown in Tables I* and II.

Since these tables are somewhat detailed, it may be of advantage to give in advance a few points by way of a very general statement of results. The time required for the 18 normal observers to pass from near to far varied between 0.50 and 1.16 seconds, a range of 132 per cent.; from far to near, between 0.39 and 0.82 second, a range of 110.3 per cent.; and from near to far and back to near, from 0.96 to 1.76 seconds, a range of 83.3 per cent. Of these observers,

* For the greater part of the results given in Table I we are indebted to M. Almack, a graduate student in our laboratory.

83⅓ per cent. required longer to change from near to far than from far to near.* If a rough classification by rank were wanted, they might readily be divided into three or more groups, with abundance of difference between groups for a graded setting of the apparatus. If, for example, three groups are chosen, fast, medium, and slow,—fast ranging between 0.96 and 1.25 seconds, medium between 1.25 and 1.55 seconds, and slow between 1.55 and 1.85 seconds,—then 28 per cent. would fall in the first class, 55 per cent. in the second class, and 17 per cent. in the third class. The observers who wore glasses all group together with the slowest of the normal class. The time from near to far for these observers ranged between 0.89 and 1.17 seconds; from far to near, between 0.41 and 0.68 second; and the time for the double excursion, between 1.48 and 1.58 seconds. The fastest of this group was 54.2 per cent. slower for the round trip than the quickest of the normal group. Under the age of thirty there seems to be no correlation in either group with the age of the observer.†

There is a possible bearing of the principles of the test on the work of the clinic which perhaps should not be ignored. That is, in the conventional acuity test accuracy alone is taken into account. No provision is made in the form of the test to include speed of performance. When speed is added to accuracy as a requirement, a degree of sensitivity is given to the test which enables a much finer grading of the resolving power of the eye to be made. For example, two eyes

* The longer time to pass from near to far may to some extent, of course, have been due to the smaller visual angle subtended by the far object. That is, the time required to discriminate the far object may have been increased by its relatively smaller angular value.

† So far but comparatively few observers have been tested above thirty years of age. The few that have been tested have averaged among the slowest of the normal group. We hope later to make a systematic study of the effect of age on speed of adjustment. In the present study our special purpose has been merely to find out whether individual differences of considerable magnitude are present well below the limit at which the influence of age might reasonably be expected to become effective.

TABLE I.—SHOWING THE INERTIA OF ADJUSTMENT OF THE EYE FOR CLEAR SEEING AT DIFFERENT DISTANCES.

Observers with Normal Eyes—Apparatus I.

Observer	Age	Degree Values			Time Values (Sec.)			Individual Differences: Deviation from Quickest				Supplementary Data			
		Near object	Near to far	Near to far and back to near	Near object	Near to far	Near to far and back to near	Near to far		Near to far and back to near		Acuity	Near-point (cm.)	Refraction	Muscles
								Seconds	Per Cent.	Seconds	Per Cent.				
Sl.	25	1	31	64	0.02	0.50	0.96	..	..	..	..	O. D.: 6/4 O. S.: 6/4	11.5 12.5	Emmetropic +.12 S. +.12 cyl. ax. 90°	Exo.
Fl.	26	1	42	73	0.02	0.68	1.05	0.18	36.0	0.09	9.4	O. D.: 6/4 O. S.: 6/4	12.5 12.5	+.50 S. +.25 cyl. ax. 180° +.625 S.	2⅔ Eso., ⅓ L. H.
St.	23	1	38	85	0.02	0.64	1.17	0.14	28.0	0.21	21.9	O. D.: 6/4 O. S.: 6/4	10.4 10.4	+.25 S. +.25 cyl. ax. 75° +.25 S. +.12 cyl. ax. 90°	2½ Eso., 1 R. H.
C.	25	1	44	103	0.02	0.72	1.37	0.22	44.0	0.41	42.7	O. D.: 6/4 O. S.: 6/4	11.3 11.3	−.25 cyl. ax. 80° −.12 cyl. ax. 95°	2 Exo., 1 R. H.
Mc.	27	1	53	105	0.02	0.77	1.41	0.27	54.0	0.45	46.9	O. D.: 6/4 O. S.: 6/4	12.5 12.5	Emmetropic Emmetropic	1 Eso.
Ln.	26	1	39	105	0.02	0.65	1.41	0.15	30.0	0.45	46.9	O. D.: 6/4 O. S.: 6/5	11.5 10.5	+.25 S. −.25 S.	1¼ Eso., 1 R. H.
F.	40	2	54	124	0.04	0.775	1.85*	0.275	55.0	0.89	92.7	O.D.: 6/4 O. S.: 6/4	15.0 15.0	+.12 cyl. ax. 120° +.12 cyl. ax. 15°	3 Eso.

* Compare this result with that obtained with the same observer with Apparatus II. It will be remembered that we have stated (p. 150) that a longer time was required for the double excursion with the first apparatus than with the second in all cases in which the determinations were made on the same observer, owing to the tendency of the eye to linger on the far object when no provision was made to cut it off just as soon as it was discriminated.

TABLE II.—SHOWING THE INERTIA OF ADJUSTMENT OF THE EYE FOR CLEAR SEEING AT DIFFERENT DISTANCES.

OBSERVERS WITH NORMAL EYES—APPARATUS II.

Observer	Age	Degree Values*				Time-Values (Seconds)				Individual Differences: Deviation from Quickest						Supplementary Data			
		Near object	Near to far	Far to near	Near to far and back to near	Near object	Near to far	Far to near	Near to far and back to near	Near to far		Far to near		Near to far and back to near		Acuity	Near-point (cm.)	Refraction	Muscles
										Seconds	Per Cent.	Seconds	Per Cent.	Seconds	Per Cent.				
H	19	1	48	44	92	0.02	0.63	0.39	1.02	..	..	..	..	..	..	O. D.: 6/4	10.3	Emmetropic	$1^1/_2$ Eso.
																O. S.: 6/4	10.3	Emmetropic	
Lz	25	1	58	48	104	0.02	0.75	0.42	1.17	0.12	19.0	0.03	7.7	0.15	14.7	O. D.: 6/4	12.5	−.12 cyl. ax. 0°	3 Eso., 1 R. H.
																O. S.: 6/4	13.0	Emmetropic	Add: Abd. =26:5
Bk.	25	1	59	55	114	0.02	0.77	0.47	1.24	0.14	22.2	0.08	20.5	0.22	21.6	O. D.: 6/4	13.0	+.12 cyl. ax. 60°	$^1/_3$ R. H.
																O. S.: 6/4	12.5	+.12 cyl. ax. 120°	Add: Abd. =22:11
L.	19	2	59	64	123	0.04	0.76	0.565	1.325	0.13	20.6	0.175	44.9	0.305	29.9	O. D.: 6/4	11.0	Emmetropic	1 Exo.
																O. S.: 6/4	8.6	−.25 cyl. ax. 5°	Add: Abd. =10: 8
Rg.	28	1	55	70	125	0.02	0.715	0.61	1.33	0.085	13.5	0.22	56.1	0.31	30.4	O. D.: 6/4 −	12.0	−.12 cyl. ax. $122^1/_2°$	$1^1/_4$ Eso.
																O. S.: 6/4 −	13.0	Emmetropic	
Ty.	24	1	55	87	142	0.02	0.69	0.76	1.45	0.06	9.5	0.37	94.9	0.43	42.2	O. D.: 6/4	12.0	Emmetropic	1 Exo.
																O. S.: 6/4	10.5	Emmetropic	Add: Abd. =22:7
D.	22	1	51	63	114*	0.02	0.825	0.675	1.50	0.195	31.0	0.285	73.1	0.48	47.1	O. D.: 6/4	9.5	−.12 cyl. ax. $157^1/_2°$	$^3/_4$ Eso.
																O. S.: 6/4	9.5	−.25 cyl. ax. 17°	Add: Abd. =18:5
S.	24	2	68	75	143	0.045	0.85	0.66	1.51	0.22	34.9	0.27	69.2	0.49	48.0	O. D.: 6/5+	10.5	−.25 S.	1 Eso.
																O. S.: 6/4	11.0	Emmetropic	Add: Abd. =14:8
F	40	1	64	81	145	0.02	0.79	0.73	1.52	0.16	25.4	0.31	87.2	0.50	49.0	O. D.: 6/4	15.0	+.12 cyl. ax. 120°	3 Eso.
																O. S.: 6/4	15.0	+.12 cyl. ax. 15°	Add: Abd. $=12:3^3/_4$
B.	19	1	56	92	149	0.02	0.705	0.82	1.525	0.075	11.9	0.43	110.3	0.505	49.5	O. D.: 6/4	9.0	−.25 cyl. ax. 150°	1 Eso., $1^1/_8$ R. H.
																O. S.: 6/4	11.0	−.25 cyl. ax. 5°	Add: Abd. =20:10
M.	18	1	79	70	149	0.02	0.94	0.635	1.575	0.31	49.2	0.245	62.8	0.555	54.4	O. D.: 6/4	8.0	Emmetropic	1 Exo.
																O. S.: 6/4	8.0	Emmetropic	
Rs.	24	2	98	60	158*	0.06	1.16	0.60	1.76	0.53	84.1	0.21	53.8	0.74	72.5	O. D.: 6/4	9.0	Emmetropic	4 L. H.
																O. S.: 6/4	10.5	−.25 cyl. ax. 90°	Add: Abd. =14:9
WITH GLASSES—APPARATUS II.																			
W.	25	3	77	12	119*	0.09	1.02	0.46	1.48	0.39	61.9	0.07	17.9	0.16	15.1	O. D.: 6/6	12.0	−.25 S. +.50 cyl. ax. 90°	$1^1/_2$ Eso.
																O. S.: 6/15	12.5	+.50 cyl. ax. 115°	Add: Abd. =20:9
Bt.	27	2	63	75	138*	0.01	0.89	0.68	1.57	0.26	41.3	0.29	74.4	0.55	53.9	O. D.: 6/5+	9.5	−3 S. −1.37 cyl. ax. 15°	Ortho.
R.	31	1	105	43	148	0.02	1.17	0.405	1.575	0.54	85.7	0.015	3.8	0.555	51.4	O. S.: 6/5+	10.5	−3 S. −1.37 cyl. ax. 165°	Add: Abd. =20:9
																O. D.: 6/4	9.0	−1 S. −.375 cyl. ax. 150°	$^1/_4$ Exo., $^1/_2$ R. H.
Hk.	27	1	82	62	144	0.02	1.015	0.565	1.58	0.385	61.1	0.175	44.9	0.56	54.9	O. S.: 6/4	10.0	−.25 S. −.25 cyl. ax. 0°	Add: Abd. =24:8
																O. D.: 6/4 −	11.0	−.50 S. −.25 cyl. ax. 120°	$^3/_4$ Eso., $^1/_2$ R. H.
																O. S.: 6/5+	10.5	−1.25 S. +.37 cyl. ax. 150°	Add: Abd. =21:9

* In the four cases marked with an asterisk, longer exposures were needed than could be gotten by changing the setting of the discs with a given position of the weights on the bar. The range of exposure needed was secured by changing the positions of the weights on the bar.

which discriminate detail within the same visual angle cannot be said to have the same acuity unless the task can be performed in the same or very nearly the same length of time; yet both might be given the same rating by the conventional test of acuity, so far as any safeguarding provision to the contrary is concerned. Indeed, when speed is made a feature of the test, differences are picked up which would be passed over entirely by the clinic test. Such a refinement of the test need not be especially cumbersome when properly applied to the needs of the practitioner, and might, it would be reasonable to suppose, be utilized to advantage as a means of making a more precise diagnosis and in checking up and deciding between corrections, at least in certain difficult and troublesome cases.

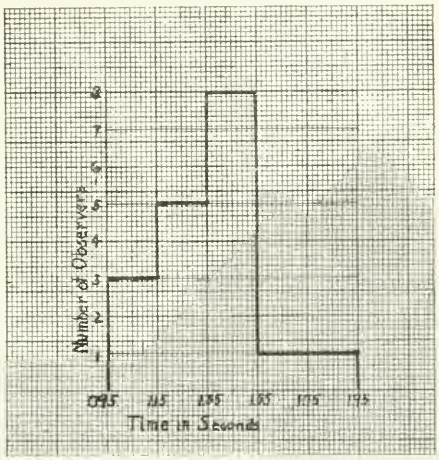


Fig. 2.—Representing the relative distribution of 18 observers graded with reference to speed of adjustment for clear seeing at different distances.

From the beginning of our work with short exposures, results were obtained which may have some interest in relation to testing for astigmatism. For example, it was found that in certain cases there was a more favorable meridian for the quick discrimination of the test object. That is, when turned into this meridian, a shorter time of exposure was needed to give the judgment required, and small deviations on either side increased the time needed to make the discrimination. In case of my own eyes, for example, a difference in result amounting to 40 per cent. was found for this

meridian and the meridian at right angles to it. A deviation of five degrees either way from this most favorable meridian gave a difference in result amounting to 16 per cent. The astigmatism was so slight that it could not be detected on the astigmatic chart. It was located by means of the ophthalmometer. A +.12 cylinder served to make the time record equal for the two meridians and to eliminate the astigmatic showing by the ophthalmometer. On further study of several cases the test was shown to possess a pronounced sensitivity to astigmatism,* even without any additional or special modification better to meet the requirements of that particular application. Some of the results of this study are shown in Table III.

TABLE III.—SHOWING A COMPARISON OF THE TIME REQUIRED TO DISCRIMINATE THE FAR OBJECT IN THE MOST FAVORABLE MERIDIAN AND THE MERIDIAN 90 DEGREES FROM THIS IN CASES OF LOW ASTIGMATISM—ALSO THE DIFFERENCE IN THE TIME REQUIRED FOR THE MOST FAVORABLE MERIDIAN AND FOR MERIDIANS 5 DEGREES ON EITHER SIDE.

Observer	Refraction	Time of Discrimination of Far Object in Seconds		Difference for Two Meridians		Difference Produced by a Change of 5° in Either Direction	
		Most favorable meridian	At 90° from most favorable meridian	Seconds	Per Cent.	Seconds	Per Cent.
R. (O. S.)	−.25 cyl. ax. 5°	0.70	0.95	0.25	35.71	0.09	12.86
R. (O. D.)	−.37 cyl. ax. 150°	0.96	1.40	0.44	45.83	..	..
F.	+.12 cyl. ax. 120°	0.80	1.12	0.32	40.00	0.14	17.50
B.	−.25 cyl. ax. 150°	0.65	1.02	0.37	56.92	0.06	9.23
T.	−1.25 cyl. ax. 170°	0.55	0.84	0.29	52.73	0.12	21.82
L.	−.25 cyl. ax. 5°	0.76	0.88	0.12	15.79	..	..

* Before the lag records are made it is recommended, of course, that astigmatisms be corrected. However, even if they are not corrected, low astigmatisms will give little trouble unless the defect is in the same meridian in both eyes. There will then be more and less favorable meridians for the quick discrimination of the test-object. The difficulty can be overcome fairly well, however, and the record be made without serious injustice to the ranking of the observer by avoiding turning the test-object into the most and least favorable meridians.

The requirements of the apparatus needed to make the foregoing determinations are comparatively simple. Some means must be provided for giving the exposures to the near and far test-objects which will immediately succeed each other in the required order, and which can be varied by very small amounts and be repeated with precision. The first of the above requirements can best be met by making the successive exposures all a part of the same system of motion. The simplest way in which all the requirements mentioned can be satisfied is perhaps to have the exposures made by a set of light-weight discs of variable open and closed sectors turned by means of a bar fastened at its center to the axle, to which the discs are attached, and provided with adjustable weights on both arms. Such a system operates as a compound pendulum and has all the characteristics and constancy of motion of a compound pendulum. The length of exposure can be varied either by changing the width of the open sector or the position of the weights on the arms. By utilizing both of these variables to their fullest extent, changes can readily be made of the order of thousandths of a second or even less, and a total range of exposure can be given varying from these values up to several seconds. All our work was done, for example, by adjusting the position of the weights on the arms to give a slow rate of turning and changing the values of the open sectors. Constancy of rate of turning, and therefore constancy of length of exposure, with a given value of open sector and a given position of weights on the bar, was secured by always releasing the bar at the same point in the arc through which it makes its swing. On the back of each set of discs is a protractor or graduated circle by means of which the values of the open sectors can be read to degrees or fractions of degrees. These values in degrees for any number of observers or observations, if the discs are made of sufficiently light material, can be converted into units of time by a single process of calibration which will be described later.

In Fig. 3 is shown the first or simpler apparatus. The exposure discs, A, B, C, and D, are cut from hard sheet aluminum, No. 20 B. & S. gauge. Each of these discs is cut as shown at X in the figure, the inner portion, radius 6.5 cm., solid; the outer portion open to a value of 172°. The breadth of this outer zone for discs A and B is 14.5 cm.; and for discs C and D, 22.5 cm. The difference in the breadth of these two sets of discs has to be such that the smaller will just cover one of the near test-objects, and the larger the other, the two objects being placed far enough apart in the same vertical plane to permit of a clear view between them with either eye of the far object. All these discs are attached to an axle to the end of which is fastened the bar, 2½ meters long, carrying the weights, M and N, which serve as the driving power of the apparatus. These weights are of equal mass, therefore the moment of turning of the system is governed, roughly speaking, by the two factors: the combined distance of the two weights from the center of rotation, and the difference in the distance of the weights from that point, provided, as already stated, the swing is always begun at the same place in the arc through which the system turns. To give stability of support, the axle turns in bearings at the ends of the two arms of a heavy Y-shaped support. A clutch, adjustable in height, supports the bar before it is released for its swing and guarantees that it always starts from the same position. The discs A and C are pinned permanently to the axle in such a position that when the bar is held in the clutch, A just covers one of the test-objects and C the other. The discs B and D are free to turn about the axle, and when adjusted for a given value of exposure are clamped in position by means of a nut and washer. Immediately in front of these discs are the two octagonal cards, along each edge of which are printed the test-letters. These E's are so turned that, by rotating the card, the letters can be presented all precisely at the same place, pointing up, down, right, left,

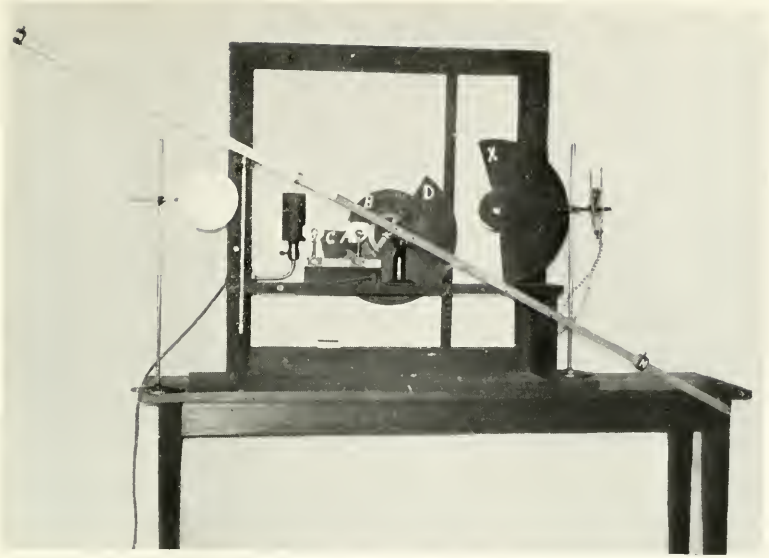


Fig. 3.

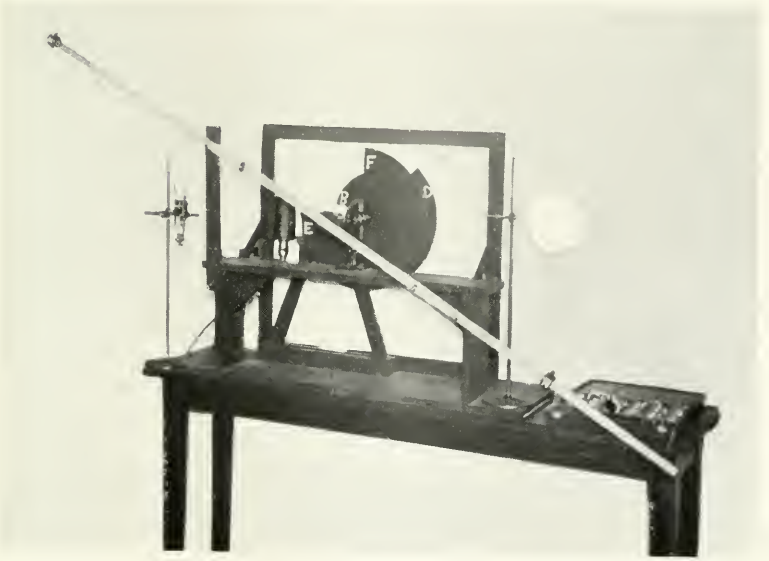


Fig. 4.

and the four corresponding 45-degree positions in any order that may be chosen. The card itself is mounted at its center on a small metal disc at the end of a grooved pin 6.5 cm. long. This pin passes through a collar provided with a set-screw, which feature permits of a certain latitude of adjustment of the distance of the test-card from the exposure discs. To provide for the rotation of the test-card this collar turns in a sleeve supported by a grooved carrier. This carrier slides on a track to permit of the needed latitude of adjustment of the test-object to right and left. The far test-object is printed on a larger circular card which is mounted at its center on a small metal disc at the end of a pin, which passes through a broad collar permitting of its free rotation. At the other end of this pin is a pulley so arranged that, by means of two cords, the card can be rotated to any position desired by the experimenter stationed at the exposure apparatus. The circumference of this card, which turns immediately behind a pointer, is graduated in degrees to indicate the meridian into which the test-letter is turned. Between the observer and the exposure discs, as near to the discs as possible, is a cardboard screen with an aperture of such a height and breadth as to give a clear view of the near and far test-object, with either eye, and to cut off the rest of the field of view and the moving discs.

The near test-cards were illuminated by diffuse light reflected from the mat surface of the back of the cardboard screen, between the observer and the exposure discs. The light was supplied by a tubular tungsten lamp inclosed in a cylindric housing provided with a vertical aperture of suitable breadth. This housing can be rotated about the lamp to give the proper angle of incidence of the light on the reflecting screen. The far test-object was illuminated by a tungsten lamp mounted in an *x*-ray deep bowl reflector so directed as to give an even illumination of the test surface and to shield the eye from glare. All of the test-objects were

brought as nearly as possible to a brightness and color match at a brightness value of 0.007 candlepower per square inch. The value of the illumination at the test-object was 5.2 foot-candles. The general illumination of the room was indirect, with an average value of 2.89 foot-candles, horizontal component; 1.11 foot-candles, vertical component; and 2.64 foot-candles, 45-degree component.

The experimental procedure was as follows: The three test-objects and the eyes of the observer were adjusted to the same vertical level, and the two near test-objects were separated far enough to give the observer a clear view of the far object with either eye. Discs A and B were adjusted so as just to permit of the discrimination of the near object immediately in front of them; and C and D, the discrimination of the far object in case the time to pass from near to far is wanted, and of both the far object and the remaining near object in case the time of the double excursion is desired. In making each determination three correct judgments out of a possible five were required. A preparatory adjustment of the observer's eye was secured by having him fixate a point on the discs in line with the near object first exposed and 3 mm. behind it. In order that the preparatory interval be as favorable as possible, the observer was required to give the signal for the release of the pendulum.

It should be noted in passing, perhaps, that the adjustment of the discs A and B is not made entirely or even primarily for determining the lag in perception with the eye in approximate adjustment for the near object, although that is an item that might be of value in our comparative study of the lag of the ocular functions of different individuals. It has been made chiefly in order that the determination of the time to pass from near to far and back again to near shall be made with greater precision. That is, it is obvious that if the observer is to begin his excursion from near to far with an exact adjustment for clear seeing at near,

it must be required as a check that he start with a task which involves a report of clear seeing at near. The mere instruction to fixate a point, for example, will not guarantee the needed adjustment. Moreover, it is equally obvious that the adjustment must be precisely controlled if the results are to be safeguarded against the variable error that has already been discussed with reference to the exposure of the far object. That is, if it is not controlled, the eye may linger too long at near or begin too soon its change toward far, and the amount of deviation in either regard may vary from time to time and from individual to individual.

In the second type of apparatus (Fig. 4) our purpose, it will be remembered, was to make it possible to do all that could be accomplished with the first apparatus, and in addition to provide for the separate determination of the time required to pass from near to far, and back again to near, in a single swing of the pendulum. In order to do this it was necessary to have behind the near test-objects a second set of discs attached to the same axle, one of the sectors of which, when properly adjusted, cuts off the far object as soon as it is discriminated. That is, in this apparatus the aperture of the two smaller sectors, A and B, of the nearer set of discs gives the time of perception of the near object on the observer's left; the aperture between B of this set and F of the farther set gives the time needed to pass from near to far; and the aperture between this disc and the disc D of the nearer set the time required to pass from far back again to near. The other discs, E of the far set and C of the near set are pinned permanently to the axle and are rigidly connected, with the edge of each at exactly the same level. Both sets of discs are provided with graduated circles. At the edge of each of the movable sectors are pointers for reading the values of the open sectors. As the apparatus now stands, the two sets of discs are both between the Y-shaped support and are only 10 cm. apart. Since the graduated circles are

on the back of each set of discs, this makes the reading of the circle on the near set somewhat inconvenient. In a new apparatus now in construction the near set will be attached between the observer and the support in which the axle turns, and the far set beyond this support. This provision will give ample space between the two sets of discs for the convenient reading of the scale on the near discs.*

With both types of apparatus, all readings are made in terms of degrees of open sector. These readings can, after any number of sets of observations, be converted into units of time by a simple process of calibration. Smoked paper is clipped to the disc across the open sector; the pendulum is released with the weights, the starting-point, etc., just as they were in the original determinations; and a time line is run across the open sector by means of an electric tuning-fork whose vibration frequency is known. The paper can be removed, shellacked, and counted at leisure. In counting, the given degree values can be laid off on the shellacked record by means of another protractor. If the discs are made of material so light that the different positions of the movable discs do not change by significant amounts the relative accelerations of the pendulum at the different points in its path,† a calibrating chart may be made once for all for the full range or any range of open sector that may be desired.

* Also in the newer form of apparatus the near test-cards are illuminated by a tubular tungsten lamp installed in the horizontal in a plane midway between the screen and the near test-objects, so that the center of its filament is about 12 cm. above the two test-objects and equidistant from them. The test-objects thus receive their light in part directly from the lamp and in part by reflection from the screen. In this way it is possible to make the intensity of light received by the two objects more nearly the same than is the case with the illuminating device shown in Figs. 3 and 4. On the platform between the two sets of discs is installed a second lamp, suitably shaded, which can be turned on and off at will for the reading of the graduated circle on the back of the set of discs nearest the observer. (Since the above paper was presented, this newer type of apparatus has been taken overseas for the purpose of studying and checking up the diurnal variations in the ocular condition of the aviators on the western front.)

† Light cardboard, for example, could be used, if desired, for these discs instead of aluminum.

Another method which we have used is to have contacts soldered to the edges of the sector, in circuit with an electromagnetic time-marker writing on the smoked paper of a kymograph. Because of a certain amount of lag in this system of recording, the method was abandoned in favor of the one described. In practical use, however, where an exact quantitative rating of performance is not required, there is no particular need of converting the readings on the scale into units of time. This is especially true if the apparatus is used, as in the Snellen method of rating acuities, merely to classify performance roughly by the rank method. That is, in this case it is set to give in turn the different levels of performance chosen, and the eye is rated by the highest level which it is able to attain.

DISCUSSION.

DR. LUCIEN HOWE, Buffalo, N. Y.: The authors of this paper deserve our thanks and congratulations for their apparent success in solving this difficult problem. Similar solutions of it have occurred to others among us, but the difficulty was to lessen the error due to the personal equation. My suggestion, for example, was to measure the time between the recognition of the near and far objects, or the reverse, by means of a key attached to a chronograph. That is very much as the astronomers measure the time when a star crosses the meridian; but the error by that method would be measured in tenths of a second, whereas Dr. Ferree has reduced this to hundredths of a second.

Dr. Ferree is fortunate in having an excellent mechanician always at hand, and his classes bring to him groups of young women with exceptional intelligence for instruction and for testing. There is no question in my mind, at least, that this and similar exact methods of measuring ocular conditions will prove important in determining the relative value of different persons in certain occupations. For example, only those who have learned how many demands are made on the aviator can appreciate how much depends on the condition of his ocular muscles, especially on the ability of his ciliary muscles to act promptly and without fatigue even

under unusual conditions. In the same way the rapid adjustment of the ciliary and other ocular muscles is almost as necessary to the sailor as is a normal color-sense. In a word, another factor in the important act of accommodation has today been made clear to us. I think American ophthalmologists are fortunate in having in at least one institution an investigator who thus contributes to our knowledge of physiologic optics.

Dr. Alexander Duane, New York City: I want to thank Dr. Ferree for his very suggestive paper, and to add one or two impressions that I gathered. First, he spoke of its practical application to aviators. Now, its application to other branches of the service seems equally obvious, and none more so than the navy. The conditions there for seeing under different circumstances and surroundings are such as call for just the kind of adjustments that he has spoken of. For example, take an officer on the bridge of a battleship in action: he has to make the quickest kind of adjustments of vision for far and near seeing of objects. There are all kinds of difficult conditions under which he has to make these instantaneous adjustments that Dr. Ferree speaks of, and it may happen that the men who might seem to be normal by our ordinary visual tests may fail because they cannot make these adjustments with sufficient rapidity. The difficulty thereby produced in recognizing signals and answering them immediately might spell disaster. A moment's delay in recognizing the turning of the ship ahead which is a comparatively short distance away, and in responding to that movement, would also spell disaster.

One other thing occurs to me: Dr. Ferree said that the question whether the speed of adjustment could be trained or not had not been determined. I believe that the ability to make these instantaneous discriminations can be trained by practice, like all these other faculties, and I think that is a very fruitful field for investigation, not only to determine whether people have these abilities or not, but, granting that they have them to a moderate degree, to enhance their ability by systematic training. That is a point that could be investigated and carried out to the great advantage of the service.

DR. C. E. FERREE, Bryn Mawr, Pa.: I believe with Dr. Duane that it should be possible to train the motor coördinations involved in the adjustment of the eye as well as other motor coördinations. In this connection it should be remembered, however, that improvement by training comes for the greater part as a practice effect for some particular or individual performance. To what extent training for a particular performance gives facility for other performances is a more difficult question to answer. At present we have no data on which to base a general conclusion with reference to the effect of training on the speed and accuracy of the ocular adjustments. The results of the observers trained in the use of the high-power microscope suggested that the point might repay investigation.

Two other points which we hope to investigate are diurnal variations and the slowing effect of age. The latter becomes of importance if men of the age of the late ex-Mayor Mitchel are to enter aviation and other vocations for which speed and accuracy of ocular adjustments are a prerequisite. The results which we have obtained thus far on older subjects (not included in the preceding tables) suggest that above the age of thirty the eye becomes not only slower in its reactions, but increasingly liable to lapses and depressions lasting through longer or shorter intervals of time, which make it a tardy and unreliable instrument for much of the work it has to do, more particularly for the special vocations requiring speed of performance. That such lapses are a menace to the aviator is now pretty generally recognized. It is a matter of common report that even the young aviator, under the strain of the prolonged performance of his task, grows stale. If so, it is obvious that a careful check should be kept on the sensory and motor foundations of his fitness for his work. In this procedure the speed of reaction of his skeletal as well as his ocular muscles doubtless should be included. In the body of the paper we have outlined a method by means of which a check may be had not only on the quickness of the motor functions of the eye, but also, by simultaneous record, those of the hands and feet and the mental functions involved in making a choice of reactions. By adding these supplementary features to the test a broader basis would be laid for keeping track of the

aviator's ability to see his objects quickly and for performing the reactions needed for the control of his machine. In discussing these subsidiary questions, however, I do not wish to obscure what we believe to be the most promising application of the test, namely, the initial grading of men as to fitness for work requiring special ocular capacities.

ON THE CONSTRUCTION OF TEST-OBJECTS, ESPECIALLY THOSE FOR MEASURING FATIGUE OF ACCOMMODATION.

LUCIEN HOWE, M.D.,
Buffalo, N. Y.

According to the plan for this meeting I was to give a description of the present form of the ergograph for measuring the fatigue of accommodation and of convergence, and also present data showing briefly the practical value of the instrument. It has been necessary, however, to change that plan. This was because—

First: The latest model, which has been in use for some time at the Medical Research Laboratory at Mineola, is about to be shipped to France. Apparently it is of assistance in classifying aviators according to their endurance of fatigue of the eyes. Without a presentation of the instrument itself, any description would be confusing.

Second: It is desirable to settle a point which was discussed at our last meeting. At that time I presented the first model of the ergograph for measuring fatigue of accommodation, but one of our members raised the question as to whether the fatigue which developed was not partly retinal rather than muscular. That led to further study, with the result that, in order to exclude even the possibility of a complicating factor, the test-letters for the near-

point have now been supplanted by test-objects of the "checker-board" pattern.

Third: The data which have gradually accumulated concerning ocular fatigue are too numerous for a single communication. Therefore it is better at present to consider only the first subdivision of the plan originally proposed, namely, the construction of test-objects, especially those for measuring fatigue of accommodation. As this means an understanding of all test-objects, it is necessary to recall for a moment the principle on which they are made and the imperfections of letters used as tests, especially for near. It may be stated here that the object of this paper is to show that black squares, arranged somewhat in checker-board fashion on a white background, or the reverse, are more accurate tests, certainly for near vision, than are letters.

In constructing any such test-objects we assume, for various reasons, that the angle of minimum vision is about one minute. Snellen and others selected letters which, when placed at the required distance, measured an angle of five minutes. That was on the supposition that each component part of a letter (for example, the projecting points on the cross bar above an L or the opening in a C) measures one-fifth of the whole letter. In this way each smallest part of a letter would be just visible.

But it soon became evident that comparatively few letters in the alphabet comply with these requirements. The question of the best form of test-letters has been the subject of special consideration by different writers, by an International Congress, and more recently attention has been called to the subject by the report of the Committee on Standardizing Test-Cards. This is published in the Transactions of the Section on Ophthalmology of the American Medical Association in 1917. Some of the imperfections of letters as tests of vision are—

First: They are not of equal value. The most recent

demonstration of that fact is given in the report of the committee just referred to. This showed that if we consider perfect vision = 1.00, then the recognition of the letter L at the standard distance would indicate V. = 0.62. The recognition of an A would mean V. = 0.71, or of a T, V. = 0.72, and so on until we reach the recognition of the letter B, which indicates V. = 1.15.

Second: Letters assist the imagination. The sight of one portion of an object which is so familiar suggests the remaining portion. If we cover the lower half of a test-line, a considerable number of the letters can still be read by an intelligent subject.

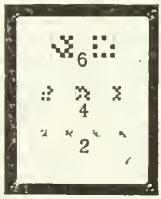
Third: When we use the letters as test-objects no effort is necessary to overcome the condition which psychophysiologists have long ago recognized as the "fluctuation" or "intermittence" of minimal visual sensations.

The meaning of that term is best understood by a simple experiment. If a person looks intently, for a short time, at an object which is properly illuminated, but so small that it subtends practically the angle of minimum vision, the retinal perception "intermits"; that is, the object seems to appear, then disappear, and after an instant it reappears. The simplest form of the experiment is to cover a bit of milk-white glass with one or two layers of thin white paper, make a minute cross or dot on the outer paper, and, illuminating it well, regard it intently.

Hering, Exner, Fechner, Helmholtz, and others have argued over the causes of this "intermittence." It is more familiar in ophthalmologic literature under the term "retinal fatigue." The practical aspect for us, as shown in an excellent monograph by Ferree, is that this intermittence of visual acuity, or retinal fatigue, occurs most distinctly when the eyes are fixed steadily on a single point. But, on the other hand, if the eyes are turned from that one point, no matter in how small a degree, to another point, then the

vision clears—the intermittence or retinal fatigue disappears. Such a change in the position of the eyes in the effort to distinguish a test-object is, therefore, an important desideratum; but no such change is necessary to distinguish a letter. We undoubtedly recognize it more by its general contour than by the recognition of each component part, which, if alone, should subtend the angle of one minute.

Other objections to the use of letters as test-objects might also be mentioned, such, for example, as the very considerable and irregular intervals in the sizes of letters in different lines. But these need not be considered here. The practical question is, what test-objects, if any, are better than letters?



Samples of "checker-board" test-object. An atypical arrangement of the squares is purposely shown. When thus photographed down for use at the extreme near-point, the print from a zinc etching gives only an idea of the original. As the ink flows, the smaller squares look like round dots.

For the usual clinical routine, perhaps it may be well for the present to retain letters as test-objects. Several reasons for this suggest themselves, such as custom, simplicity, and rapidity.

Also, the characters which have thus far been suggested as a substitute for letters have not been generally adopted, in spite of some excellent reasons in their favor.

But we could use another character or group of characters as test-objects which are free from the imperfections already mentioned as inherent in letters. It is to these characters that I wish to direct attention. They consist of nothing more than black squares arranged somewhat in checker-board fashion on a white background. When placed at the required distance, each integral square, whether black or white, subtends an angle of one minute. If, therefore, with 25 of these small black and white squares (five on each side) we construct one large square of checker-board fashion, we have the elements

which should exist in the ideal test-letter. In practice, however, evidently a checker-board which always retains five black and white squares in one direction and five in the other direction is useless because the subject will immediately see that each group is composed of a total of 25 small squares. To count them would be unnecessary. This objection is easily obviated by making the number of black and white squares considerably less than 25 and by arranging them irregularly.

The advantages of the checker-board tests drawn to scale are:

First: The test-object can be uniform as compared with others made on the same plan, but of different size.

Second: The recognition of one part does not suggest the other part—that is, if the subject counts a certain number of small component squares, that does not suggest the number which remains uncounted.

Third, the most important of all: The factor of retinal fatigue is apparently quite eliminated. In counting the component squares in each chart, the axes of vision must pass from one such square to another.

Theoretically, then, this appears to be the best test both for distance and near.

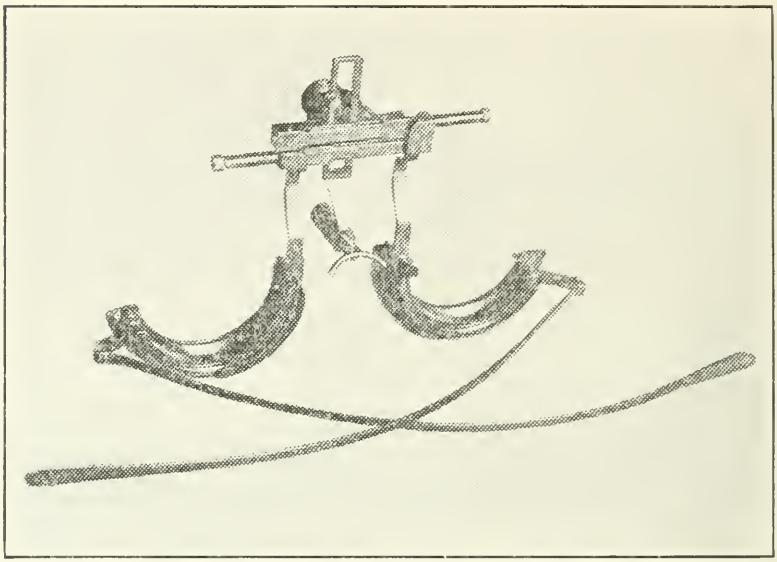
I am not prepared to say as yet that this checker-board pattern is so far superior to letters that we should discard letters as tests in clinical work. That is a question for future determination. I am certain, however, that test-objects which are made on this plan are better than the letters which have heretofore been used for measuring fatigue of accommodation.

THE TRIAL-FRAME FOR TEST-LENSES, WITH DEMONSTRATION OF A NEW MODEL.

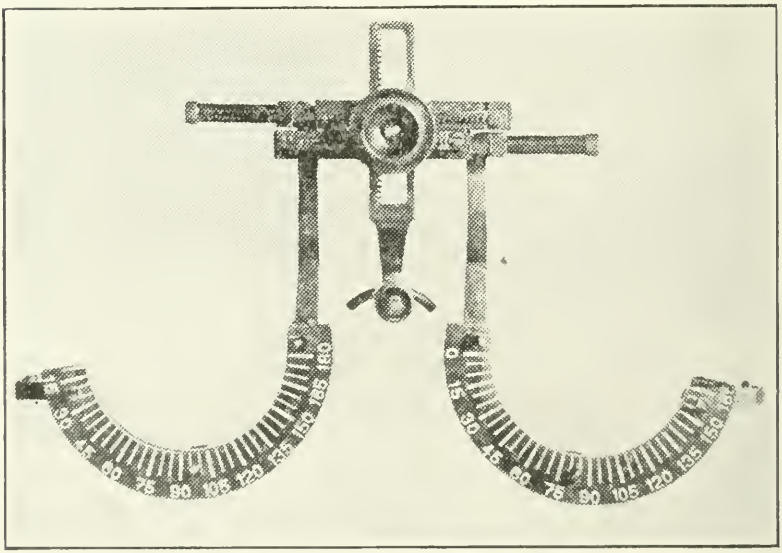
WALTER L. PYLE, M.D.,
Philadelphia.

The ordinary trial-frames and test-lenses seldom conform to the modern standards of the skilful medical refractionist. In hospitals and dispensaries they are especially faulty and often ludicrous. It is most important that the trial-frame shall be firmly and snugly placed on the sides of the nose and adjacent cheeks, in approximately the position in which the prospective spectacles or eye-glasses will be worn. In this respect all frames fixed to mechanical instruments, such as optometers, are at fault, often clumsy, and are signally deficient for accurate testing.

The prime requisites for a trial-frame are: rigidity, lightness, proper position on the face, and ease of general adjustment and manipulation of the trial-lenses. There is also a necessity for individual adjustment of the two sides, to permit perfect binocular centering. The test-lenses, carefully centered, should be placed before the eyes, properly inclined, and just clearing the eyelashes. The bridge of the frame should not wound the skin or cause irritation or pain. Rotation of the cylindric lenses must be easy, silent, and unnoticeable, to prevent even momentary diversion of the patient's attention. The frames should be grooved and slotted to allow rotation of the cylinders at least 180 degrees. The cells with the cylinders should have short handles; and the rotation should be effected by light contact on these handles with the tip of the first finger. It is



Model of trial-frame. Back view.



Model of trial-frame. Front view.

impossible to practise noiseless and smooth rotation when using cells without handles. The placing of the trial-lenses in the spring clips is awkward, disconcerting to the patient, and trying to the oculist. Only by the use of a properly grooved and slotted trial-frame can the ideal examination of the refraction be made.

For many years I employed the best types of the standard trial-frames, with minor modifications suggested by Dr. George M. Gould and myself. For the last six years I have used one of three frames made under the direction of Dr. Gould. Generally speaking, this has been satisfactory, but fails to permit bilateral individual adjustment. I showed a modification of this frame last year at the meeting of the A. M. A., but it lacked the necessary central universal adjustment.

The trial-frame which I present now has a universal vertical and horizontal adjustment by rack and pinion movement, permitting adaptation for any peculiar facial conformation. This adjustment is placed in the center of the frame. The frame is grooved for two lenses. The front groove, for the cylinders, is slotted to allow rotation of the cells with handles. A third test-lens may be placed in front of the frame, resting on three small hooks.

The bilateral independent adjustment is effected by two double screws operated on the outer upper sides of the frame. The frame is placed on the face, quickly set by the universal adjustment, and supplemented when necessary by the movement of the independent double-screw adjustment, until the lenses are properly centered.

Broad shell coverings on the inner sides of the two cell-holders permit the snug application of the frame to the sides of the nose and cheeks. The nose-piece proper is only used to fix and maintain the proper position.

The graduated semicircles are enameled in dull black, with distinct white lines and figures, allowing the maximum ease in reading the axes. The whole frame is finished dark and dull, to prevent annoying reflections.

AN IMPROVED SYSTEM OF ILLUMINATION FOR THE ELECTRIC OPHTHALMOSCOPE.

CARL KOLLER, M.D.,
New York.

The credit for having conceived and executed the idea of an electric ophthalmoscope belongs to Wm. S. Dennett. The advantages of such over the old-style instrument are many, and so obvious that it is not necessary to relate them. The chief advantage, however, which gives it such a superiority and makes it an instrument with which the unexperienced may explore with ease the background of the eye, is rarely touched upon, and certainly not generally understood by many who use it.

In the old-style ophthalmoscope the picture of a flame is thrown on the background, where it can be seen, and where it illuminates and makes visible a small area. To explore a larger part of the fundus the picture has to be moved from place to place, and the ensemble of the picture has to be pieced together in the imagination like a mosaic. A large part of the background which could be seen if it were only illuminated is dark. Its size depends on a number of factors, of which the width of the subject's pupil and the closeness of approach of the observer's eye are the most important. Of this potentially visible "field of vision" only that part becomes actually visible which is illuminated by the image of the flame on the retina. To the limitation described there exists one single exception. When the picture of the flame falls on the disc of the optic nerve, the light is dispersed in the opaque fibers of the nerve, and the

whole nerve becomes visible, be it ever so much larger than the picture of the flame.

If it were possible to produce on the background an illuminated area as large or nearly as large as the field of vision, a considerable part of the whole background could be taken in with one glance. Such a possibility theoretically exists. If we could concentrate the rays emanating from the light source into a "real image" located at the second nodal point of the eye (that is, a little forward of the posterior pole of the lens, about 8 mm. from the apex of the cornea), practically the whole interior of the eye would be illuminated, and the illuminated area would be larger than the field of vision. As a fact, this arrangement is realized even with the old-style ophthalmoscope, when the "indirect method of examination with the inverted image" is being used. But to its realization with the "upright image" there are peculiar difficulties. Many years ago I tried, and I am sure many others have tried before me, to realize this condition by using very strong concave mirrors, but the reflexes produced on the cornea are so troublesome that the background cannot be seen satisfactorily.

The advent of the electric ophthalmoscope changed all this. A comparatively tiny light source could be utilized, and its real image thrown in the desired place. Practically in all the models the image of a small electric lamp concealed in the handle is thrown into the eye by means of a mirror inclined at an angle of 45 degrees, or, which is the same, by the hypotenuse of an isosceles rectangular prism. The upper edge of the mirror or prism reaches up to the line halving the aperture, and throws light in through the lower half of the pupil, while the observer looks through the upper part of the pupil. In the model of the late Dr. Marple the mirror reached higher up, but was dented in the middle, corresponding with the axis, thus allowing the observer to look parallel with the course of the rays.

It will be noted that in all the devices one chief condition compatible with practical construction is that the direction of the rays emanating from the lamp, and that of the rays entering the subject's pupil, be perpendicular to each other.

Both the mirror and the prism devices have the fault that they set a certain limit to the approach of the two eyes, and thus materially contract the possible field of vision, for the plane of the mirror or the reflecting hypotenuse of the prism slopes from the subject down to the observer. A canal, as it were, is interposed between the two eyes, preventing near approach, which is the chief contributing factor to a large field of vision in direct ophthalmoscopy.

Several ways to eliminate this fault were discussed with Mr. R. Wappler, of the Wappler Electric Mfg. Co., some time in 1910 and 1911, and under my guidance, trying to materialize the correct principles of an ophthalmoscope, Mr. Wappler constructed this model, which has been in my possession and in daily use since 1912. It has one distinguishing feature over all the other models in its illuminating device.

A prism with its sharp edge up is placed before the aperture, overlapping the lower half. The upper angle is about 38 degrees, the angles at the base are 90 degrees and 52 degrees, or approximately so, varying with the index of the glass. The anterior surface slopes down toward the subject. A pencil of light entering the base of such a prism is reflected in it twice—first on the anterior (hypotenuse) surface, and then a second time on the posterior silvered surface. Ultimately it emerges from the anterior surface at right angles to its original direction, which was along the stem of the instrument. The prism is not necessarily rectangular; the posterior surface may slope a little toward the observer. A great variety of prisms can be constructed which will emit light at right angles to the direction of its entry.

The base of the prism is ground convex, so that the pencil

of light is made to converge to a point about 20 mm. in front of the prism. This brings a real image of the filament in the neighborhood of the nodal point, from whence the rays diverge again, illuminating a large part of the background with an even light. The arrangement of the prism, sharp edge up, allows a very near approach to the subject's eye, thus insuring a large field of vision.

This model is far from perfect in other respects: I hope to be able to improve on it. My present object was to show the advantages of this system of illumination.

DISCUSSION.

DR. WALTER E. LAMBERT, New York: I should like to ask Dr. Koller how the illumination of his electric ophthalmoscope differs from that of Dr. May's.

DR. CARL KOLLER, New York: That is easily answered; there is no difference whatever. The illuminating system used in the instrument sold as the "May ophthalmoscope" is the one which was worked out by myself and Mr. R. Wappler. It has been put into that instrument without my knowledge or consent.

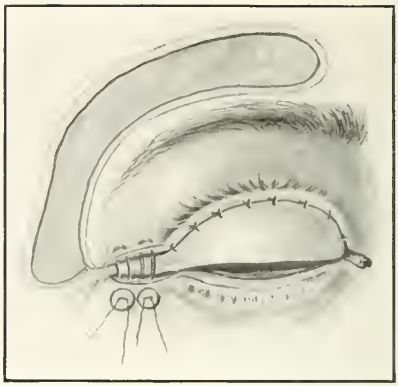
DR. WALTER L. PYLE, Philadelphia: One of the greatest advantages of the electric ophthalmoscope is the ability of the observer to approach close to the patient's eye. Unfortunately, most of these instruments have a direct lens-rotation, such as in the Loring form. The operation of such is not nearly so satisfactory as the model with the rotation indirect by a wheel below. With the latter it is possible almost to get the electric ophthalmoscope on the cornea. I have had fitted to my electric model the ophthalmoscope of my own suggestion. This instrument, like the models of Knapp, Harlan, and Keyser, has the lower wheel, and is the smallest made. The optical manufacturers seem to be utterly oblivious to the advantages of having the closer position obtained when the finger is not turning the wheel of lenses directly and pushing the instrument away from the face.

SOME OBSERVATIONS ON THE RESTORATION OF THE ORBITAL SOCKET.

P. N. K. SCHWENK, M.D., AND WM. CAMPBELL POSEY, M.D.,
Philadelphia, Pa.

The restoration of a socket of sufficient depth and of such configuration that an artificial eye of suitable shape may be properly fitted into it is never an easy task, particularly when the occlusion of the socket is complete and no conjunctival lining of the lids remains. It is not the purpose of this paper to enumerate all the methods which have been devised by operators for the accomplishment of this purpose, or to enter into comparison with the excellent results obtained by Weeks and others by the employment of Wolfe and Thiersch grafts. These operations are well known and properly appreciated. The ease, however, with which the lower culdesac may be restored by the ingenious transposition of the skin of the cheek subjacent to the lid, into the floor of the orbit, as practised by Maxwell, led the writers to a combination of this method with a utilization of a skin-flap taken from some point above the orbit for the formation of the upper culdesac. Zentmayer has successfully utilized Maxwell's procedure in forming the upper culdesac, but this operation is complicated by the presence of the levator, and is not as easy of performance, nor are the results as satisfactory, as when applied to the lower lid. All who have practised plastic surgery about the orbit have experienced the rapidity with which healing occurs in this region, and the adaptability and vitality of flaps transplanted from parts adjoining. An exact planning of the incisions, and a precise coaptation of the margins of the flaps to the tissues with

A



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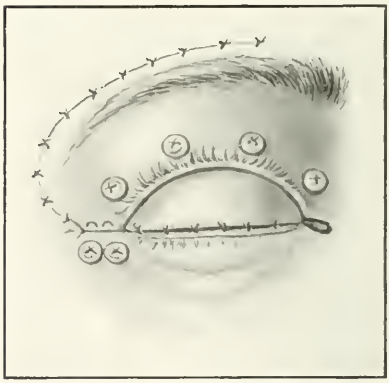


D

Fig. 1.—A, Division of external canthus and separation of the lid margins; B, outline of pedicled flap; C, transposition of flap from forehead into upper portion of culdesac, and restoration of external canthus; D, coaptation of flap margins to parts adjoining.

which they are brought in contact, insure prompt healing, and after a time an often undetectable scarring.

Our method of procedure is as follows: A careful separation of the margins of the two lids from each other and a free dissection of the tissues underlying. Then follows a division of the external canthus. The lower culdesac is then restored by Maxwell's method and the upper by transposing a long flap taken from the skin of the forehead above the brow, care being taken, however, that no hairs of the brow are included in the margin of the flap (Fig. 1). The edges of the flap must be nicely coaptated to the margin of the upper lid, and the upper border of the skin secured by the Maxwell procedure. The next step is of great importance, namely, the restoration of the external canthus, for unless this be accomplished at this time, the conformer which is introduced between the lids at the completion of the operation will not be retained in the culdesac, and a subsequent operation for the restoration of the canthus will be necessary. To secure a firm canthus, two double-needled sutures of No. 4 silk are passed through the skin of the upper pillar of the old canthus and brought out through the raw surface of the pillar; they are again reunited into the raw surface of the lower pillar, and made to issue upon the skin surface of that structure. They are then finally tied on two pearl buttons. The pedicle of the flap is thus buried under this bridge of tissue, and unless the tension upon the pillars is too great, which may be avoided by a careful dissection of the tissues of the canthal pillars prior to inserting the sutures, union is prompt and satisfactory. Should the tissues give way, the canthus may be restored later, after the flaps have united, by excising a diamond-shaped area of skin from the pedicle of the flap and uniting the edges of the denuded area in the manner just described.

Some of our colleagues have criticized our method of canthus repair, saying that a sinus would result from the bury-

ing of the skin-flap under the bridge formed by the union of the two pillars. As a matter of fact, however, this has not occurred, and in the three cases about to be reported union was firm and there was no trace of a fistulous tract. Nor were we bothered by outgrowth of hairs from the flap. This may probably be accounted for by the youth of the patients and by the absence of hair from the tissues whence the flaps were derived. During the first few months there was considerable desquamation of skin from the flaps, but this gradually subsided and the sockets became dry and more and more commodious. A point to be accentuated is the importance of retaining the conformer in the socket for a long time, interfering with it as little as possible. The conformer being of the same form as an artificial eye, gradually molds the orbit into the shape necessary for the retention of the prosthesis. A firm bandage should be kept constantly applied over the orbit for at least two weeks. The stitches inside the culdesac should be removed at the end of a week —those from the canthus in eight or ten days.

CASE 1.—J. A., aged twenty-four years, applied to the Wills Hospital for the restoration of the left socket, which had been obliterated as the result of a lime burn, as a consequence of which the burned globe had been removed some years previously. Examination showed complete ankyloblepharon, and it was impossible to insert even the smallest probe between the firmly united lids. The skin of the lids and the surrounding tissues was uninjured. The patient was etherized, and after the lids had been carefully dissected apart, the external cavities divided, and the underlying tissues freely undermined, the lower culdesac was restored after the method of Maxwell. A long, pedicled flap was then obtained from the forehead above the brow, carefully sewed into position in the upper part of the wound, and a conformer inserted into the socket thus obtained. The canthus was then restored in the manner already described and a bandage applied (Fig. 2).

Healing was prompt. As the patient lived at a distance,

Fig. 2.—Case 1. Restoration of canthus.

Fig. 3.—Case 4. Before operation.

Fig. 4.—Case 4. After operation.

he was permitted to go home, with the conformer in excellent position. Unfortunately, however, the oculist to whom he was referred, not appreciating the importance of careful oversight over the patient, permitted him to become careless in the flushing out of the socket with boric acid solution, in consequence of which the nasal end of the conformer slipped out of the socket and a rapid contraction of the newly formed cavity resulted. Upon his return to us, it was found that the nasal sulci of both the upper and lower culdesacs had contracted, and the conformer was no longer retained under the pillars at the outer canthus. Subcutaneous incisions were made under local anesthesia along the lid borders and under the tissues at the canthus, and double armed sutures buried in the apices of the culdesacs, being brought out upon the skin surface and tied over buttons, about 4 or 5 mm. from the ciliary borders of the lids. The conformer was again reinserted and the patient kept under close observation for a month. At the expiration of this period a small segment of skin was excised from the somewhat redundant base of the flap, where the pedicle had been twisted in turning the flap into the orbit. An artificial eye can now be worn with comfort and the cosmetic result, apart from the immobility of the eye, is perfect.

CASE 2.—R. B., male, aged twenty-one years, right eye and conjunctival culdesac badly burned by lime in November, 1917. After enucleation, dense cicatricial bands almost entirely obliterated the socket, the inner portion of the lower culdesac alone having any appreciable depth.

Ether was administered, and the lower culdesac restored by Maxwell's method. Some weeks later the external canthus was divided under ether, the upper lid freed from its cicatricial connections by dissection, and a flap which was taken from the skin of the upper lid just below the brow superimposed upon the raw surface thus obtained. The canthus was restored by the method just cited after a conformer had been inserted into the amply spacious socket. On the fifth day, however, the stitches holding the pillars of the canthus in apposition gave way, necessitating a refreshening of the edges of the flap and a reunion of the pillars some time later. Union was permanent, and a suitable sized eye is now worn with comfort.

Case 3.—Male, aged eighteen years, lost his right eye from a lacerating wound, demanding enucleation. In consequence of faulty technic in this operation, the upper lid was firmly bound down to the underlying tissues by dense cicatricial bands. The lower culdesac was also somewhat contracted.

Ether was administered, the external canthus divided, and the lid freed of its cicatricial connections by dissection, the conjunctiva being undermined uniformly to the margin of the lower lid. The lower culdesac was then formed by the method which will presently be reported, leaving a raw surface in the upper half of the socket. This was covered by a large pedicled flap taken from the skin of the upper lid just below the brow, as in the preceding case. After careful union of the edges of the flap to the surrounding tissues, and the apex of the culdesac deepened by three double mattress sutures, which were passed through the skin-flap about 4 mm. from the lid margin and brought out through the skin of the brow about 6 mm. from the ciliary border, a conformer was inserted and the canthus reëstablished as in the two previous cases. The raw surface marking the site of the flap was carefully covered by uniting the edges of the wound, and a firm dressing applied. Recovery was uneventful, and a proper-sized eye was retained without difficulty.

In the opinion of the authors the Maxwell operation is admiraby adapted for the restoration of the lower culdesac, as repeated trial has proved. The one objection to the procedure, however, is the tendency that the lid evinces to ectropionize at its outer half after healing has occurred. This may be partially overcome by making the flap narrower by 2 or 3 mm. than recommended by Maxwell, especially at its outer portion. The authors have not experienced the puckering in the skin below the lid, mentioned by some writers, except in one instance, where the deformity was readily overcome by excising a small wedge-shaped portion of skin, with the base up, at the point of greatest skin redundancy. As the operation seems to be but little practised in this country, the authors have thought it might be of interest to describe the various steps.

Maxwell's Operation.

Instruments.—Scalpel, dissecting forceps, pressure forceps, glass mask, needles, and needle-holder.

First Stage.—An incision is made at the bottom of the shallow culdesac in its whole length, some 5 or 6 mm. deep. It is made the same depth along all its extent (*a–b* in the diagram).

Second Stage.—On the skin of the lid are made two incisions, one about one-sixth of an inch below the lid-margin and the other curved below this, enclosing between them a semilunar area of skin about 12 mm. broad in the widest part. The length of this curved incision (*c–d* in the diagram) must be greater than that of the first. The crescent of skin thus marked out is used to form the lining of the new culdesac.

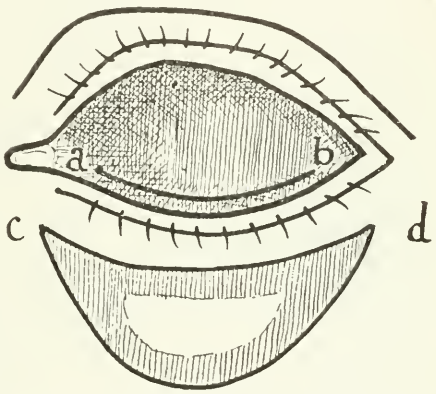


Fig. 5.—Incisions for the Maxwell operation.

Third Stage.—The upper limiting incision of the crescent is deepened until it joins the incision in the culdesac. The margin of the lower lid now forms a bridge attached only by its two extremities.

Fourth Stage.—The crescent of skin is dissected up from its borders until only a relatively small part (about one-sixth of the whole) near the center remains attached to the subjacent tissues. This is represented as unshaded in the diagram.

Fifth Stage.—The bridge of skin bearing the lashes is held forward, and the crescent slipped under. The two extremities are secured into the angles of the first incision, and to its posterior lip the upper border of the crescent is fastened by two or three sutures. The lower border of the crescent is then brought up and stitched to the conjunctival edge of the bridge. The crescent is thus doubled on itself, and forms

a groove, the posterior wall of which is made by the upper, the anterior by the lower, half of crescent. The undissected pedicle holds the floor of the groove down.

Sixth Stage.—It remains to close the wound on the surface of the lid, and this must be done carefully or there will be a troublesome scar. The two edges are very disproportionate in size: the lower much longer than the other. The first step toward closing the wound must be a stitch in the center of both edges, then one on each side, midway between the center

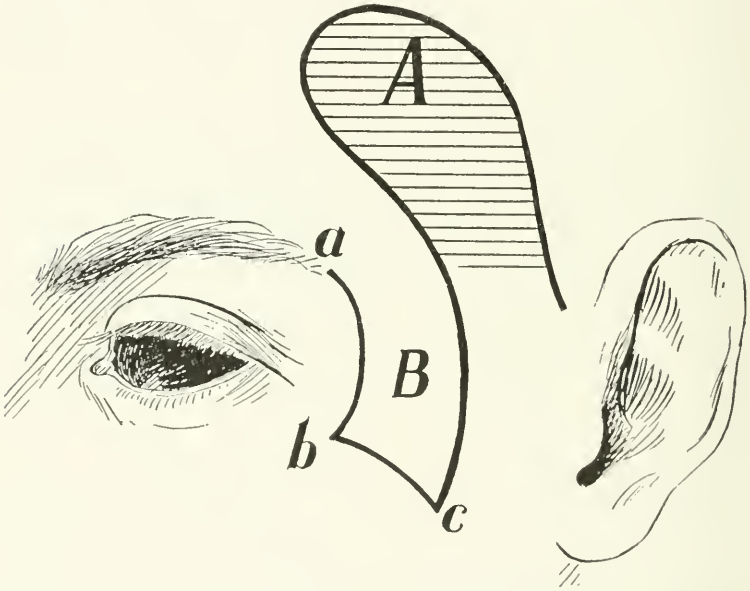


Fig. 6.—Incisions for the Golovine operation.

of the extremities. The coaptation of these two edges is the most difficult part of the operation.

This completes the whole procedure, but, before the pad and bandage are applied, a glass mask, preferably with a central opening, must be placed within the lids to depress the new-formed culdesac until healing is advanced. It should be left in for a week or ten days: the central opening is an advantage, as it enables the socket to be washed out easily.

Another ingenious method for restoration of the socket has

been proposed by Golovine. In cases of exenteration of the orbit this writer endeavored to supply a socket suitable for the reception of an artificial eye, by slipping a tongue of skin taken from the temple under a bridge of undisturbed skin at the outer aspect of the orbit. His plan of procedure was as follows: A cutaneous flap *A* is first shaped from the temple, as shown in Fig. 6. A semicircular incision, *a–b*, is made and continued to *c*, through a subdermal opening that com-

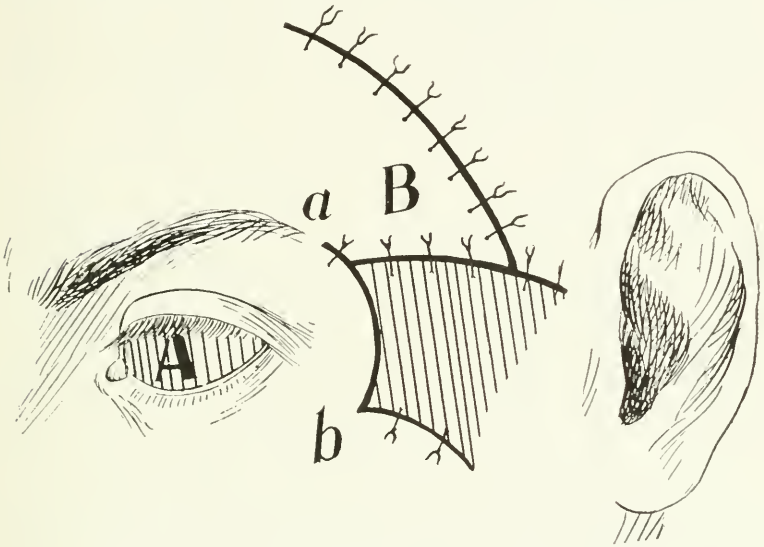


Fig. 7.—Closure of the skin incisions, Golovine operation.

municates with the orbit along *a–b*, the upper rounded edge of the large flap is slipped under, and the remaining tissues rearranged as in Fig. 7. The sliding flap applied and arranged within the orbit (epithelial surface outward) is carefully stitched to the cut edges of the remaining conjunctiva, and so a space is formed in which the prosthesis may rest.

In cases where the occlusion of the socket is incomplete, some of the conjunctiva being still present, flap transplantation is not always necessary and other methods are preferable.

Wiener's method is especially applicable to this class of cases, as the authors can affirm, after its successful trial in several instances. As some years have elapsed since its publication, the various steps are given in Dr. Wiener's own words, as follows:

"My method has its use where the socket, while shrunken, has still a small amount of conjunctiva left. An incision (*A*, *B*, *C*, Fig. 8) is made through the conjunctiva, and a flap, including only the conjunctiva, is carefully dissected down to the lid margin (*A*, *C*, Fig. 8). This dissection, after being started with a knife, can easily and quickly be finished with a small curved blunt scissors. The dissection is then continued with the scissors so as to loosen the skin below

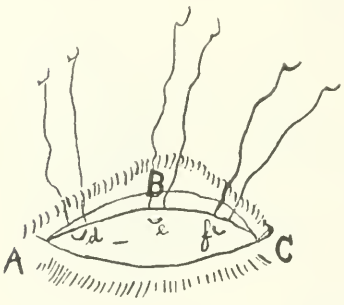


Fig. 8.

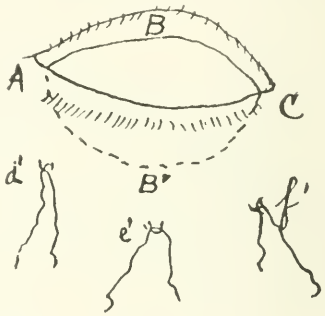


Fig. 9.

Wiener operation.

the lid margin (*A*, *C* to *B*, Fig. 9), leaving a raw surface toward the bulbar side extended from *B* to B^1 and *A* to *C* (Fig. 9), and on the palpebral side *A*, *B*, *C* (Fig. 9). Then sutures with a needle on either end are introduced at the points *d*, *e*, *f* (Fig. 8), and passing them through the bottom of the newly made sulcus are brought through the skin at *d'*, *e'*, *f'* (Fig. 9) and tied over a button. This gives a conjunctival covering for the lower lid and leaves the bulbar surface *A*, *B*, *C*, *B'* (Fig. 9) to be covered. This is done by covering a lead plate, previously shaped, with grafts from the thigh, and placing carefully in position. A dressing is then applied and both eyes bandaged, the patient remaining undisturbed in bed for four days, when the outside dressings are replaced by clean ones without disturbing the plate. On

about the eighth day the plate may be removed, cleaned, and replaced. The glass eye can usually be worn by the tenth or fourteenth day. The stitches at d', e', f' (Fig. 9) are not removed, but are tightened each day after the fourth day until they pull through. This is an additional help to holding the lower sulcus intact, as the internal scars resulting tend constantly to pull on the bottom of the sulcus and thus heighten the effect. It is important that these stitches should be allowed to pull through and not be removed before, for one is tempted to remove them after a week, when the lower sulcus may appear too deep. I have made this mistake, thinking I had secured a lower sulcus of exaggerated depth, when, on removing the stitches, it rose too high and became almost obliterated, so that the stitches had to be replaced."

In 1915 one of the authors of this paper (Dr. Schwenk), without knowledge of Dr. Wiener's contribution, in an attempt to restore a shrunken socket, where some mucous membrane was still present, proceeded as follows:

The conjunctiva was undermined and brought forward, doubled upon itself to form a new culdesac. To do this the conjunctiva was entirely loosened from the floor of the orbit from a point just below the margin of the lower lid to a point 3 or 4 mm. above the nerve-head, by means of scissors, curved on the flat, entrance being made at the outer canthus. A transverse band of skin about 6 mm. wide, having its upper limits 3 mm. from the lid margin, was undermined from canthus to canthus by means of a Graefe knife inserted at the temporal side. By means of the scissors the remaining tissues of the lid were then cut through transversely into this subcutaneous pocket at a point 3 mm. from the lid margin, thus increasing the depth of the orbital cavity by an amount equal to the depth of the subcutaneous dissection. Three double-armed heavy silk mattress sutures were inserted in the loosened conjunctiva from above at a point 7 mm. from its attachment at the lid margin, and the needles were carried into the subcutaneous cavity, where they were passed

out through the skin at its lower part and tied on Ziegler pearl buttons; a gold conformer was then inserted.

Considerable hemorrhage resulted from cutting the hypertrophied conjunctiva, but there was no subcutaneous ecchymosis, and healing resulted without undue reaction. The stitches were allowed to remain seven days, when the conformer was replaced by a glass eye.

The operation is indicated in cases in which the conjunctiva is sufficiently redundant to allow of its being slipped into the anterior cavity. It differs from the Wiener procedure in the location of the culdesac anterior to the cartilage of the lid, instead of posterior to it. In the Wiener operation a large denuded area from which the conjunctiva has been taken is left exposed in the socket, which must be covered with Wolfe or Thiersch grafts. In the author's method no raw surfaces remained, as all the dissection is done beneath skin and conjunctiva. The following case is reported as illustrative of this method.

Case 4.—M. L., male, aged twenty-eight years. Right eye had been enucleated eleven years before on account of severe injury in a bottling establishment. Badly fitting cheap glass eyes were worn for many years, until cicatricial contraction of socket prevented their retention. Examination showed the conjunctiva to be much thickened, corrugated, and filled with cicatrices (Fig. 3).

Operation under ether as follows: By means of slightly curved-on-the-flat scissors the conjunctiva was entered at the outer canthus and two-thirds of the orbital mucous membrane was undermined, cutting cicatrices for some distance above the apex of the orbit and anteriorly to the margin of lower lid. Hemorrhage was pronounced, but was controlled by applications of adrenalin. A Graefe knife was then inserted just below outer canthus, and a broad band of skin was loosened and undermined, about 3 mm. from margin of the lid and 3 mm. wide parallel to its edge, across the entire width of lower lid. The submucous and the subcutaneous cavities were then united by subcutaneous incision across

whole lid. Three double armed silk mattress sutures were then inserted into the loosened conjunctiva 6 mm. from lid margin, the conjunctiva lifted from its bed on the floor of the orbit, and carried with needles to the apex of subcutaneous cavity and passed through the skin externally and each tied over a pearl button to avoid puckering of the skin. The conjunctiva was carefully adapted to the lid and floor of orbit and stitched to orbital tissue to avoid sliding or displacement. The stitches thus inserted served as anchoring points and encouraged union. A gold conformer was inserted, and a firm dressing was applied. The stitches were removed on the eighth day. Recovery was uneventful, and the result all that could be desired, a proper sized prosthesis being worn with comfort. Fig. 4 shows the condition two and a half years after operation.

DISCUSSION.

Dr. William Tarun, Baltimore, Md.: I want to indorse what Dr. Posey has said of the advantages of the Maxwell operation. Ten years ago, shortly after the description of the operation appeared, I had an opportunity to employ it in an appropriate case, and was impressed by the result secured. The procedure is especially applicable where the conjunctiva is somewhat contracted, and where the artificial eye has produced scar tissue in the lower culdesac, with an eversion of the lower lid. If care is taken to attach the flap to the lower margin of the dorsal plate, the ectropion disappears and the lid comes into proper position. At the same time it produces a large sac in the lower part, and also inversion of the lid, which corrects the deformity. The operation has, to me, a distinct advantage over any other, particularly in conditions of the lower lid. I have not had an opportunity to try it in the upper lid, but I feel that the levator muscle would be a factor in influencing the result obtained.

Dr. William Zentmayer, Philadelphia: Dr. Tarun spoke of doing the operation on the upper lid. I have done this and found that the levator offered no hindrance to perfect technic. In this particular procedure the levator must be cut and secured by a suture. The skin-flap is then slipped through the incision into the orbit, which is readily accomplished. When you do a Kuhnt-Heisrath, the incision made

through the tarsus disturbs the levator fully as much, but ptosis does not result. I have done the operation for the restoration of the lower culdesac five or six times, and the results have all been favorable. I agree that the mistake that is apt to be made is to make too broad a flap. Eight millimeters is sufficiently wide. You need only a narrow sulcus. I saw a case recently in which I had operated on both culdesacs four or five years ago. The result was still good.

In the operation of Dr. Weeks, satisfactory as it usually is, the shrinking of the flap may give an imperfect result with the lapse of time; that, at least, was my experience in the one case in which I did it.

Dr. Edward B. Heckel, Pittsburgh, Pa.: It is not only the difficulty with which these operations are done, but also the difficulty in arriving at a successful conclusion. I have had the opportunity of operating on several cases in the last ten years, and used a one-piece autogenous epithelial graft to restore the socket in partial or complete destruction. In one case the upper part of the socket was destroyed by a burn. By dissection the socket was enlarged, and a one-piece epithelial graft was taken from the inner surface of the arm. The conformer consisted of a piece of wet cotton sufficient to fill the socket. This was covered with a piece of rubber dam, tied in front with strong silk, and not left open. The external canthus was not slit. The one-piece epithelial graft was laid over the conformer, with the epithelial side against the rubber tissue. The lids were pulled down, and a compress bandage applied quite firmly. If there is no odor or discomfort, the bandage is allowed to remain six or seven days. When the dressings are removed, and the conformer taken out, the socket is as large as the conformer. The parts are irrigated daily, and a small amount of bichlorid vaselin, 1:3000, applied to the socket. I have had no graft that has not taken. The excess of graft shrivels up and disappears.

Something has been said about obtaining a graft without hair upon it. I find that in taking a graft it is almost impossible not to have some hair and sweat-glands upon it. I operated on a case in which the entire socket was destroyed. I took the skin from the inner surface of the arm, and found, after a while, that there was a growth of hair and odor from

the socket, as a result of the sweat-glands that were in the graft. The glands have degenerated and disappeared, and so have the hair-follicles. The transplantation of a few hairs and sweat-glands appears not to be objectionable.

Dr. R. A. Reeve, Toronto, Can.: I think that Dr. Heckel is to be congratulated upon anticipating a line of treatment that is in vogue in France and Great Britain. It was spoken of by Sir Arbuthnot Lane on his recent visit. He said that they restore the orbital cavity in very serious injuries by making an incision, more or less deep, and taking, as the doctor did, the epithelial graft and applying it as he has done. They simply employ the plastic material that dentists use for making casts, select an appropriate size, apply the graft on it, and stitch over it so that the whole thing is tight. The explanation of the losses of transferred flaps that we sometimes regret so much is that you do not keep the surface everywhere in contact with the raw underlying surface. By stitching the whole flap in tightly you get complete contact. In from five to ten days, upon removal of the gutta-percha, there is a perfect cavity. In addition, if one requires to resort to any measure to provide for the use of an artificial eye, it is only necessary to make an incision and utilize a piece of cartilage, properly shaped, stitching it in under cover.

Dr. William H. Wilder, Chicago, Ill.: I have had the opportunity of doing considerable of this kind of work at the Illinois Eye and Ear Infirmary. I have used, for holding grafts firmly in the socket, plates of lead, tin, or the gutta-percha material that dentists use, which can be molded to fit the culdesac that has been made and to hold the flap in place. The method originated with the late Dr. Hotz, and had been adapted and modified in various ways. Cases of total symblepharon of the orbit, and also cases of symblepharon where the eyeball is present, are suitable for such treatment; but I have found, in almost every case in which I ever attempted it, that a Thiersch graft, or a modified Thiersch graft, was as valuable as a Wolfe graft. I call a modified Thiersch graft one in which not only the epithelial layer of the skin but also the firmer tissue of the corium is taken without going into the subcutaneous tissue. The chief

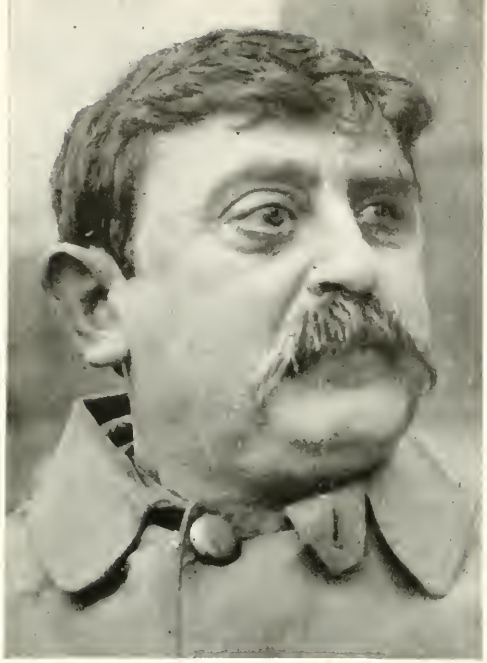
objection to the Wolfe graft is the difficulty of dissecting away the subcutaneous and the fatty tissue. With the modified Thiersch graft one gets a graft of good firmness. In preparing the socket for the plate, scar tissue may have to be dissected away, but the socket is made deep enough above and below to hold a plate that is fashioned out of lead or tin or gutta-percha. This plate can then be covered with paraffin of a high melting-point, with which it can be built up to any degree desired to fill the socket. After cutting off the Thiersch graft from whatever site selected, it can be transferred directly from the razor to the plate, over which it is carefully folded so that the raw surface will be outward. The graft adheres to the plate, which can then be introduced into the socket prepared for it. In forty-eight hours it can be removed, the socket cleansed, and the plate carefully replaced. It should be worn for a week or ten days until the socket is ready for the artificial eye. The technic of this method I have presented in previous communications.

REPORT OF FOUR CASES OF ORBITAL TUMORS SUCCESSFULLY REMOVED, WITH PRESERVATION OF VISION, THROUGH AN ORBITAL INCISION ALONG THE EXTERNAL ORBITAL RIM.

WM. CAMPBELL POSEY, M.D.,
Philadelphia.

CASE 1.—Male, aged forty-eight years. A continuous and gradual proptosis of the right eye past six years. No pain in globe, but occasional temporal, supraorbital, and frontal headache. No history of trauma or inflammation. Health good. Wassermann negative.

Examination.—Right eye proptosed forward 14 mm. in advance of its fellow, with downward displacement of about 5 mm. The globe gives the impression of being about to pop out of the orbit. No irregularity of orbital rim, no palpable mass. No pulsation. Globe cannot be pushed back into orbit. Slight bulbar injection; anterior segment of globe otherwise negative. Pupil, 3 mm., reacts to light and



Case 1.—Before operation.

Case 1.—After operation.

Case 2.—Before operation.

Case 2.—After operation.

on accommodation. Moderate neuroretinitis. Left eye negative. V. R. E. $=\frac{6}{9}$; L. E. $=\frac{6}{6}$. *X*-ray examination of orbit and surrounding sinuses negative. Nasal examination and transillumination of the sinuses negative.

Operation.—Ether. Incision over outer rim of orbit, as directed by Krönlein. After dissection of the tissues a large encapsulated tumor, much the shape and size of the eyeball, found posterior to the eye and superior to the optic nerve, but without connection with either structure. Mass removed through the incision which was extended superiorly to give room for the exit of the tumor mass. Healing prompt, although for some days there was marked proptosis of the eyeball forward in consequence of the hemorrhage occasioned by the trauma of the operation. Two months later proptosis reduced to 4 mm. Some ptosis and restriction of the outward movements of the eye. Both of these conditions are, however, rapidly subsiding. Vision as before operation. Some subsidence in the neuroretinitis.

Pathologic Report.—Macroscopically the specimen consisted of a hard, oval-shaped mass, measuring 22 x 35 mm. in its longest diameter. The external surface was smooth. On section, it was encapsulated. The tissue was white in color, resembling smooth muscle. Microscopically the tumor was encapsulated, the capsule consisting of encircling layers of long spindle-cells arranged compactly in a manner resembling smooth muscle. The interior of the tumor was composed of the same type of tissue, less compactly arranged, with a tendency to the formation of whorls. The blood-vessels were typical, and in most part were in the center of the whorls above mentioned. Diagnosis, spindle-cell sarcoma.

CASE 2.—Male, aged twenty-nine years. Gradual protrusion of right eye past five years. No trauma. No inflammatory symptoms.

Examination.—Right eye proptosed directly forward 10 mm. in advance of its fellow. Health good. No traumatism. No pulsation. Impossible to press globe back into orbit. No orbital mass palpable. Globe negative. *X*-ray and nasal examination of orbit and accessory sinuses negative. Wassermann negative. V. R. E. $=\frac{5}{7.5}$; L. E. $=\frac{5}{5}$.

Operation.—Ether. Incision as for Krönlein procedure. Large encapsulated tumor resting upon optic nerve below found immediately behind globe without connection with adjoining structure. Readily removed by blunt dissection. Healing prompt. At end of three months proptosis entirely gone; ocular movements free in all directions. Scar over wound almost invisible.

Pathologic Examination.—Macroscopically the specimen consisted of a round mass measuring 24 mm. in diameter. It was spongy to the touch. The center consisted of a network of fibrous tissue. Microscopically the specimen was composed throughout of dense fibrous tissue, most of which was arranged in a crinkly manner, and was devoid of nuclei. The arrangement was loose, presenting many open spaces, which were lined in some instances by a very thin endothelium. The growth was spongy, the sponginess being attributed to numerous open spaces within the aforementioned loosely arranged fibrous tissue. Diagnosis, fibroma.

Case 3.—Female, aged twenty-seven years. Right eye prominent since early childhood, but no apparent increase in past ten years. Health good. Wassermann negative.

Examination.—Right eye proptosed 8 mm. in advance of its fellow. Smooth, hard mass, readily palpated at upper, outer angle of orbit, extending downward as far as outer commissure. V. R. E. = $\frac{5}{5}$; L. E. = $\frac{5}{5}$. Incision made over mass at outer angle of orbit. Cyst with firm capsule and dermoid contents removed without difficulty. Recovery rapid. At end of month proptosis almost entirely disappeared; some ptosis and limitation of external motion of eye.

Pathologic Examination.—Dermoid cyst.

Case 4.—Female, aged eighteen years. Gradual protrusion of left eye, without inflammatory symptoms, for past eight months; this condition being attributable to an attack of typhoid fever three years previously, as a sequel of which the posteervical and submaxillary glands of the left side remained permanently swollen. One gland ruptured spontaneously. General health good. Family history negative for tuberculosis. Has never had diplopia, but covers left eye when reading to avoid seeing same line twice.

Examination.—Left eye proptosed down and somewhat out, 7 mm. in advance of its fellow. Under the upper lid at the outer third a firm, movable mass, somewhat tender on firm pressure, is readily palpable, which gives the impression of being slightly lobulated and of a glandular nature. V. R. E. $= \frac{6}{6}$; L. E. $= \frac{6}{15}$. The posterior border of left sternomastoid muscle is lined with glands varying in size from a pin to a small hickory-nut. Similar glands are situated in the submaxillary region. Some suggestion of enlargement of middle lobe of thyroid. Von Pirquet and Wassermann negative. Moderate anemia. *X*-ray and nasal examination negative.

Operation.—Ether. Incision made along and below left supraorbital ridge at the outer part, immediately over the palpable mass. Tumor the size of a large almond and surrounded by a firm capsule exposed and removed by blunt dissection, permitting eyeball to recede into its normal position. Healing prompt. At end of a month, all proptosis had disappeared and the movements of the eye were normal.

Pathologic Examination.—Macroscopically the specimen consisted of a hard, oval-shaped mass measuring 28 by 15 mm. in its longest diameter. It was encapsulated. On section, tissue resembled that of a gland, the center of which was hard and fibrous. Microscopically the growth was encapsulated, the capsule contributing to the formation of trabeculæ, which permeated throughout. The structure was that of an adenoma, the acini of which were small, each acinus being surrounded by a very definite connective-tissue framework. The acini were about 70 microns in diameter. Near the capsule the acini arrangement was lost, probably due to compression in this region by the firm capsule. Diagnosis, adenoma.

With the exception of the fourth case, where the tumor was palpable and where the incision was naturally made directly over the mass, the precise location and the character of the neoplasm occasioning the proptosis were in doubt. The degree of the proptosis present suggested a tumor of considerable size in each instance, and the character of the displace-

ment seemed to locate the growth in the posterior portion of the orbit. Under such circumstances it seemed advisable to enter the orbit through its outer part and to place the incision in such a way that, if found desirable, the entire wall of the orbit might be resected after the manner of Krönlein, thereby affording easy access to the orbital apex, and permitting the easier removal of a growth from that region. Fortunately, however, in none of these cases was this found necessary. The author has performed the Krönlein procedure upon six cases, the notes of several of these having been submitted to this Society at previous meetings. All these cases terminated successfully, and in only one was there any appreciable scarring, namely, in that of a child five years of age with a large dermoid cyst of the orbit, where the detached fragment of bone broke down, leaving for a time a discharging fistula.

All those who have performed the Krönlein operation, however, confess to the difficulty they encountered in displacing the wedge-shaped fragment of bone, resected from the outer angle of the orbit, *in toto*. On account of the contour of the orbital ridge and the contents of the orbit, it is impossible to employ straight saws with advantage. When chisels are employed, the bone is apt to split, and it is practically impossible to remove the apex of the wedge as recommended by the author of the operation. In several of his cases the writer has first drilled the bone with a dental engine and then employed the chisel, but even with this modification the apex of the triangle of bone has remained. Some years ago he was much impressed by an article by Magitot and Landrieu, of Paris, translated by Ziegler, of Philadelphia, which contained what appeared to be a valuable modification of the Krönlein method. As the description of this method may have escaped the attention of some of the members of this Society, it may not be amiss to quote it *in extenso*. As the authors follow the original Krönlein

method precisely until the resection of the bone is reached, it will be necessary to detail their observations only from that point. They are as follows:

Now we proceed to the bony sections. This is without doubt the most delicate stage. The reason for this is on account of the extreme hardness of the orbital border. The bony tissue of this region is certainly the most resistant of the body, and its eburnation in aged subjects is normal. It is not necessary to depend upon chisels in order to finish the operation. Fortunately, we have saws. But straight saws are inconvenient. Using them horizontally, they no sooner enter the bony tissue than they injure the soft parts: for on one side are the muscles of the skin and on the other side are the eyeball and the eyelids.

In order that the bony block shall reach as deep as the pterygomaxillary fissure, it is necessary that the first section shall start from this fissure, passing from the depths to the surface. Therefore, every method which commences by cutting into the orbital border only succeeds in detaching the edge of the orbit, that is to say, only a small part of the external wall.

The orbital periosteum having been detached as far as the pterygomaxillary fissure, a malleable retractor is introduced into the cavity, pressing back the periosteum and the eyeball toward the nasal side. The bony border having been equally exposed by a separator (rugine), the temporal aponeurosis is incised. The object is to reach the pterygomaxillary fissure and introduce into this orifice a wire saw which should emerge through the orbit. Therefore, in order to do this, one should open up the temporal fossa a little in order to permit the passage of a probe or bodkin. With the aid of a grooved director one should then detach the whole exterior surface of the orbital apophysis of the malar. Above all, the operator should bear in mind that it is in the angle formed by the malar apophysis and the zygoma (a great deal lower than is generally believed) that the probe must be introduced in order to penetrate into the orbit through the pterygomaxillary fissure. It is important at this point to press backward the muscular mass. It should be held back tightly by a retractor intrusted to an assistant.

Of course, one should avoid detaching the exterior periosteum of the temporomalar, because it is through this membrane that the bony flap, dislocated and put back in place, is nourished. Then, taking a flexible bodkin, or if it is necessary a grooved silver canula, or better, a malleable conductor, the surgeon should give it a somewhat curved form and fasten to it the wire saw of Gigli. It is necessary then to recognize the location of the pterygomaxillary fissure. This fissure, concealed by fatty tissue, should be easy to locate with the end of a blunt instrument. It is well to note that the operator always has a tendency to look too high for it. It should be borne in mind that it is to be found at about 2.5 cm. from the orbital border formed by the *body* of the malar.

The pterygomaxillary fissure being found, the operator should introduce into it the flexible bodkin until he sees the point of the instrument emerge into the orbit, seizing it with a hemostatic forceps and drawing it toward him. He should then attach and introduce into the fissure the wire saw which he then disengages from the conductor.

There is nothing more to do now but to resect the orbital wall. The incision should be made sufficiently low in order to cut off the base of the orbital apophysis of the malar. The section, being easy, is made in a few seconds by the drill-bow movements imparted to the saw, which the two hands of the operator must hold *tightly stretched* in order to avoid kinks or breaking.

The section being finished, the surgeon searches at the extremity of the orbital apophysis of the malar for *the suture which unites the malar to the temporal*, the location being quite visible. Having recognized it, he marks it with a protected saw, or, in its absence, a very small straight saw, but the depth of this groove should not exceed 5 mm. The saw is then laid aside and the operator takes a small graver-chisel which he holds vertically, placing the cutting edge of it in the groove made by the saw. All that is necessary now is a simple blow of the mallet administered very moderately on the chisel in order to detach the *whole orbital apophysis of the malar* as far as the pterygomaxillary fissure.

The bony block being liberated without splintering is then *displaced* without difficulty, and ultimately *put back in place* at the end of the operation.

Although the writer has practised this method upon the cadaver, he regrets that the opportunity has not arisen to perform it upon the living subject. Although a number of cases of orbital tumor have presented themselves, the growths were removed in all with the preservation of the globe without the necessity of resecting the bone; indeed, it would appear, from the author's experience, that this step is necessary only in cases of tumor of the optic nerve, or in tumors situated at the apex of the orbit, which are of such magnitude that they cannot be forced between the globe and the external wall of the orbit without injury to the former. While the modification of the Krönlein method suggested by these authors seems rational and feasible, the author confesses to having encountered great difficulty in inserting the Gigli saw through the pterygomaxillary fissure into the orbit. It is not easy to deflect the temporal muscle sufficiently to expose the fissure, and the angle at which the aneurism needle must be made to enter the fissure is an awkward one. It is possible, however, to follow the steps outlined by the authors on the cadaver, and it may be easier, as oftentimes happens in other operative procedures, to perform it upon the living subject. The writer hopes that this method will be given a trial by others.

REPORT OF TWO ORBITAL TUMORS.

A. EDWARD DAVIS, M.D.,
New York.

CASE 1.—Endothelioma Affecting the Orbit and Frontal Bone.—Mr. C. C., aged nineteen years. Family history negative; is robust—in fact, a football player. Consulted me December 21, 1917, because of swelling of the lids and prominence of the right eye, the swelling extending from the eyebrow to the hair, circular in shape, of an elevation one-

half inch, and of bony hardness; no pain; temperature and pulse normal. Patient thinks the swelling on the right side of the forehead may have resulted from injury while playing football, although the skin surface was never broken. V. R. E. = 20/30; L. E. = 20/20, not improved; J. 1, 6 to 18 inches each. Slight swelling of nerve-head in right, with the veins slightly enlarged; fundus normal otherwise. Tn. Blind spot normal in size, also the fields and pupils. Slight restriction in movement of the eye upward and outward, and double vision obtained when he looks far upward and outward.

January 4, 1916: Through an incision one and one-half inches long, just below the upper rim of the orbit, the lacrimal gland and part of the roof of the orbit were removed. Pathologic examination of the specimen showed it to be an endothelioma. One week later the roof of the orbit and 1½ inches of the frontal bone to the hair on the forehead, together with the meninges, extending itself into the brain tissue, were removed. The patient made a good recovery, so far without return of the trouble. After the wound had firmly healed the patient has been and is now under *x*-ray treatment at week intervals.

Present condition: The patient is in perfect health and at his work as a clerk, and feels no ill effects from his operations. There is no sign of brain hernia, the right side of the forehead being on a level with but not protruding beyond the left, the symmetry of the two sides being remarkable. The advisability of a bone-graft has been considered, but, as the opening in the skull is so large, it has been questioned if the space could be successfully bridged over by this method of procedure; so far only a protective cap has been used. There is slight ptosis present, and all the external muscles of the eye are inactive save the obliques, both of which function. There is a divergence of 20 degrees. The pupil is normal in size and reaction, and the accommodation the same as in the sound eye. Vision in each eye same as before operation; simple binocular vision is absent. The fields are normal. No attempt has as yet been made to remedy the ptosis or place the eye in a straight position, and will not until the *x*-ray treatment is completed, and a return of the growth considered unlikely.

As to the pathogenesis of endotheliomata, Parsons* states that "The exact nature of endotheliomata and their exact position amongst the sarcomata have not been settled." He himself classifies them as fibrosarcomata. The prognosis in such cases is rather favorable, as they are not nearly so malignant as the round-cell type of sarcoma.

CASE 2.—Myxoglioma of the Optic Nerve and Orbit, Intradural, being the recurrence of a Spindle-celled Sarcoma(?), Intradural, removed seven years previously by the Krönlein Method.—The previous history of this patient has been kindly furnished to me by Dr. Julius Wolf, of this city, and is herewith given:

"November 20, 1910: R. D., aged eight years, no history of previous illness; the third of healthy children. Three weeks ago the left eye began to look strange to the parents and teacher, and since then the eye has been steadily growing worse. At present it shows a well-marked exophthalmos directly forward; yesterday there was no convergence; today the left eye is markedly convergent, the inner margin being behind the caruncle. The pupils react normally. The upward movement of the eye is 2 mm. above the horizontal plane. The movement of the eye extends to within 2 mm. of the external canthus. The ophthalmoscope shows a marked choked disc in the left eye. Both eyes are hypermetropic about 4 D. with some astigmatism. The lacrimal gland cannot be felt. There is a decided feeling of resiliency of the left eyeball when pushed into the orbit.

"November 21st: The internal strabismus has disappeared; the exophthalmos has not changed any.

"November 23d: Convergence present again; V. L. E. = 15/50, disc still choked. *X*-ray by Dr. Caldwell shows blurring in the ethmoid reaching to the orbit. Diagnosis: tumor of the orbit. Operated by the Krönlein method, and removed part of the optic nerve, which had a spindle-shaped tumor inside the optic nerve sheath, about one inch long by three-fourths in thickness at center. On examination of the tumor a diagnosis of spindle-celled sarcoma was made."

This girl first came under my care at the Post-Graduate

* Parsons: Pathology of the Eye, vol. ii, p. 707.

Hospital six months after the operation by Dr. Wolf. The condition found was convergence of the left eye 30 degrees, due to section of the external rectus muscle; V. = 0; post-neuritic atrophy of the optic nerve; pupil normal in size and reacted consensually. There was a discharging sinus at the lower end of the Krönlein incision, which had healed except at this point; patient was in good health, and there was no sign of metastases.

The fistulous opening was enlarged, some dead bone removed, followed by immediate healing. The patient remained well until the spring of 1918, having been put under observation by myself every few months all this time—about seven years. During March of this year the patient noticed that the eye was again becoming prominent. The eye is now displaced 4½ mm. forward and slightly inward. No pain is complained of. The ophthalmoscope shows a white optic disc, but no inflammatory symptoms in the fundus. Pupil normal in size and to consensual reaction. *X*-ray shows the apex of the orbit up to the eyeball filled with a growth. Because of the recurrence, a complete exenteration of the orbit was advised.

On June 13th, under ether narcosis, Dr. Hubbard, my chief of clinic, performed a complete exenteration, first enucleating the eyeball, then removing the entire orbital contents, including periosteum. The optic nerve could be seen both at the anterior portion and at the posterior portion of the tumor, so it is evident that not all the optic nerve was removed at the first operation; in fact, the retinal circulation was maintained in the eye up until the time of the second operation.

The pathologic examination of the tumor is as follows:

Macroscopic.—The eyeball has increased tension. The pupil measures 2 mm. The iris is white and thickened. On section the lens is much flattened and has an opaque, greenish-yellow nucleus. The vitreous is fairly normal. The retina is transparent, and the choroid shows loss of pigment, especially at the equator. There seems to be linear radial thickening near the papilla. The capsule of Tenon is hemorrhagic, and the optic nerve is enlarged to 2.5 cm. in diameter for a distance of 4 cm. behind the bulb.

This tumor is resilient, and its cut surface seems to show a hypertrophy of the optic nerve, somewhat soft and degenerating. The growth is surrounded by muscles, gland, and fat.

Microscopic.—Cross-section of tumor of optic nerve reveals a picture entirely of neuroglia cells and fibrils. All nerve-fiber elements have disappeared, and their place is filled with myxoid material through which the neuroglia tissue is intertwined with scant remnants of the old connective-tissue septa. Irregular hyaline bodies are frequent, and an occasional amyloid concretion. The sheath is not remarkable.

Diagnosis.—Myxoglioma.

Parsons* states that the term "glioma" has been used loosely for any connective-tissue growth occurring in nervous structures, and cannot be considered authentic unless the growth is proved to consist essentially of a neuroglial hyperplasia by the successful application of specific neuroglial stains.

I may say that, besides Dr. Meeker, of the Post-Graduate Laboratory, Professor Ewen, of Cornell, has examined some of the sections of this tumor, and has pronounced it myxoglioma. The variety and rarity of such an intradural growth certainly make it worth while to report. That a glioma should succeed a sarcoma, if the first growth was a sarcoma, is also of interest.

* Parsons: Pathology of the Eye, p. 699.

TUMOR OF THE ORBIT, WITH EXTENSION TO FRONTAL AND TEMPORAL REGIONS, TREATED BY *X*-RAYS.

WILLIAM M. SWEET, M.D.,
Philadelphia.

The following history of a case of orbital tumor does not differ in its essential features from the usual type of these growths. The case is reported as an evidence of the value of the Roentgen rays in controlling the disease for several years, as shown by the bone regeneration that followed the treatment:

S. H., aged twenty-six years, first noticed swelling of the eyelids of the right side two years prior to the date of examination, May, 1908. In a few months the sight of the right eye began to fail, and there was slight protrusion of the globe. There was no pain, and the symptoms remained unchanged until six months ago, since which time the prominence of the eye increased. Well-developed, robust, healthy individual, with normal urinary and blood tests, and with no family history of malignant disease. No history of injury. Right eyeball turned down and slightly outward, and protruding 8 mm. beyond the level of the left eye. Outward rotation impaired, but fair rotation in other directions. Palpation showed a hardened mass above eyeball, extending from near the inner canthus to slightly beyond center of orbital margin. Pressure failed to relieve the exophthalmos. The right pupil was 4.5 mm., reacted sluggishly; the left 3.5 mm., reacted freely. The left eye was normal in all respects. The optic disc of the right eye was pale, the veins full and tortuous, particularly the smaller twigs, and the arteries reduced in caliber. Visual field contracted on temporal side.

Following a request of the patient to endeavor to save

the eyeball, an incision was made through the brow, the orbital fascia separated, and the finger passed into the orbit. A dense, hard mass was found attached to the upper and inner orbital wall, not connected to the eyeball or optic nerve, but extending some distance beyond the globe. In order to remove the mass the eyeball was enucleated, an incision made through the external canthus, and complete exenteration of the orbital tissues made.

After separating the periosteum with the tumor it was found that the disease had attacked the bone in the upper nasal portion of the orbit, and there was an opening large enough to admit the finger through the bone and dura. The orbit was packed, and healing was uneventful. *X*-ray treatment of the orbit was given by Dr. W. F. Manges, of Philadelphia, and continued later by Dr. George C. Johnston, of Pittsburgh, in which latter city the patient was in business.

Three years after the operation swelling was noticed of the frontal bone, particularly in the outer portion. The *x*-ray treatment was directed to this region, and later also to the temporal bone, which showed enlargement. These measures held the disease in check, and enabled the man to continue his work. A set of stereoscopic *x*-ray plates showed the right frontal sinus completely occluded by the disease, with extensive involvement of the frontal and temporal bones. These plates indicated, as reported by Dr. Manges, that healing was in progress in the frontal region, the process being one of bone regeneration, but in the temporal region there was not the same evidence of bone reproduction, indicating activity of the disease in this situation. The *x*-ray treatments were given every four weeks, the full therapeutic dose being administered for five minutes in different directions to reach the diseased areas.

Late in 1916 the man died. Until three days preceding death he was comparatively comfortable. He then had intense headache, followed by delirium. No postmortem examination was permitted. The tumor was shown to be an endothelioma.

DISCUSSION OF PAPERS OF DRS. POSEY, DAVIS, AND SWEET.

Dr. J. H. Claiborne, New York: The operation of Krönlein is very popular, and it gives an opportunity to those eye surgeons who have knowledge of general surgery to exploit themselves, but I am convinced it is not necessary in the majority of cases of tumor of the orbit. Most tumors can be removed by the operation through the brow if the incision is carried well beyond the orbit. I removed a cylindroma of the orbit an inch or more in length, about the size, in fact, of a large olive, which was situated in the orbit just behind the lacrimal gland. The latter was attached to it. The incision was made through the brow, well out, and the orbit was exposed from one end of the incision to the other. This gave a free play to the finger. With the back of a scalpel careful dissection was made and the little finger introduced well toward the nasal side of the tumor; it was removed in fifteen minutes from the time the knife was taken in hand. I found it necessary, however, to shave off a little of the outer and upper part of the orbital ridge. The thing was done so easily that I suggested in a paper in which I described the operation that a small section of the outer and upper orbital ridge be removed, leaving the periosteum, and that this be sewed together so as to induce the reproduction of bone-cells. Dr. Gifford, of Omaha, later performed and suggested a similar operation, but subsequently wrote me that after he had described it he had read the paper in which I had made the suggestion. I do not believe the Krönlein operation is necessary in all the cases in which it is done, and, indeed, it ought to be done only in very large tumors or those which would have to be removed piecemeal.

Dr. R. A. Reeve, Toronto, Can.: I should like to ask Dr. Davis why, after an interval of a week, he did a secondary operation. His was a case of endothelioma. A good many years ago, by a large incision extending through the lower part of the brow from the external to the internal margin of the orbit, I was able to remove a sarcoma of the lacrimal gland which was extending along the roof of the orbit. I simply peeled off the periosteum of the roof of the orbit, removing, of course, the gland itself. The patient lived for

two years, at least, and she died from some acute disease which could have no connection with secondary development from the sarcoma.

Dr. Davis spoke of there being no recurrence for, I think, twelve years. I reported, some years ago, before this Society, a so-called recurrence of sarcoma in the orbit fourteen years after having enucleated the eye which contained the original sarcoma; and I expressed the opinion that it was not, strictly speaking, a case of recurrence. If a man lives to the age of fifty-six without the occurrence of sarcoma, and the view is correct that it is a development of the embryonic tissue, why should not this occur in another part without its being a recurrence, when the man is fourteen years older? I do not think that we should always speak of a recurrence of the growth. I think that one can actually circumscribe the diseased area, and have an appearance *ab origine* at another site, exclusive of what may be fairly termed secondary or metastatic growths.

Dr. Wm. Campbell Posey, Philadelphia: Dr. Claiborne has intimated that the Krönlein operation should be limited to disseminated growths throughout the orbit, whereas to my mind such cases call for exenteration, especially if they are malignant. Dr. Claiborne speaks of sewing up the periosteum as though it were a simple matter. It has been our experience, on the other hand, that this layer is rarely as tangible as one might imagine, and sewing would not often be possible.

I would also say that after removal of the contents of the orbit we have found fulguration a valuable means in checking for a time further extension of the growth.

Dr. A. Edward Davis, New York City: I should like to answer Dr. Reeve's question regarding a secondary operation in Case 1. I did that because I considered an endothelioma to be a malignant growth belonging to the sarcomatous group. As soon as the nature of the growth was ascertained, Dr. Judd performed the major operation and removed the growth in the forehead, which was an inch and a half in diameter, involved the meninges of the brain, and extended into the brain itself. That is why we did the secondary operation so soon.

As regards Case 2: Besides the rarity of the growth (glioma of the optic nerve), the chief point of interest is that glioma should follow sarcoma. The question is whether we could get a glioma following a sarcoma.

DR. CLAIBORNE: In reply to Dr. Posey, I would say that my suggestion concerning the Krönlein operation in large tumors and scattered tumors, such as diffuse sarcoma, was based upon a case of large sarcoma of the orbit which was removed by Dr. Pooley piecemeal through the brow section. He attempted to remove all the tumor, but naturally did not, and finally sewed the wound up. The patient, who was a very old man, made a good recovery. The proptosis almost disappeared and the patient lived for several years. I am not certain whether he ultimately died of the sarcoma or not, but it struck me at the time that a Krönlein operation would have furnished a still better result. This case convinced me that possibly it is not always necessary to remove the eye and exenterate the orbit, even in a diffuse sarcoma.

OPHTHALMOSCOPIC CONDITIONS SIMULATING SARCOMA OF THE CHOROID.

(*a*) GRANULOMA OF THE CHOROID. (*b*) TUMOR FORMATION ASSOCIATED WITH RETINITIS WITH MASSIVE EXUDATION.

HARRY FRIEDENWALD, M.D.,
Baltimore, Md.

The diagnosis of choroidal sarcoma may be difficult. The tumor may be hidden in front of the ophthalmoscopic field or behind a detached retina. But when a neoplasm is clearly seen within the eyeball by means of the ophthalmoscope, the only question which ordinarily arises is whether it is choroidal sarcoma or a retinal glioma. Other forms of neoplasm are of such rare occurrence that they are scarcely ever taken into consideration; and the differentiation between sarcoma and glioma is ordinarily determined by the

age of the patient, as well as by the difference in the appearance of the growths.

In reporting the following cases, I believe that I am presenting conditions of unusual rarity, the character of which could be definitely established only by pathologic examination.

CASE 1.—The patient, a young girl, aged eleven years, had been under the care of Dr. Louis P. Hamburger since the beginning of 1908, and consulted him because of headache. The diagnosis was neurasthenia. Her family history was negative. The patient had had measles, chickenpox, and whooping-cough. She was a girl of small build, fairly well nourished. There were occasional choreiform movements about the head, mouth, and eyelids. A complete examination was negative in all other respects. In spite of the treatment the condition remained the same, and the chorea became more active at times. During 1914 she was seen by Dr. Hamburger a number of times because of the constant headache, and in December she was under the care of Dr. Sidney Cone for scoliosis.

The patient was first seen by me in November, 1900, when she was brought for a slight blepharitis. I saw her again June, 1904, at which time she complained that the eyes pained her when reading; both eyes had perfect vision and a very low degree of hyperopia. In November, 1907, she was again examined, and her glasses were changed. At this time an examination was made under atropin and her hyperopia was found to be 1.50 D. in each eye, with perfect vision.

In January and February, 1911, she was again examined and again the same hyperopia was found. The right eye showed, sph. +1.50, V. = 6/4, almost perfect; left eye sph. +1.50⊃cyl. −0.50 axis 120°, V. = 6/4, almost perfect. The left glass was changed to correspond with this examination.

In January, 1912, another examination was made because of eyeache and of blurred vision, but I found perfect vision in each eye with the correcting glasses.

On March 7, 1915 (her age was then eighteen), she again consulted me and complained of circles and flashes of light

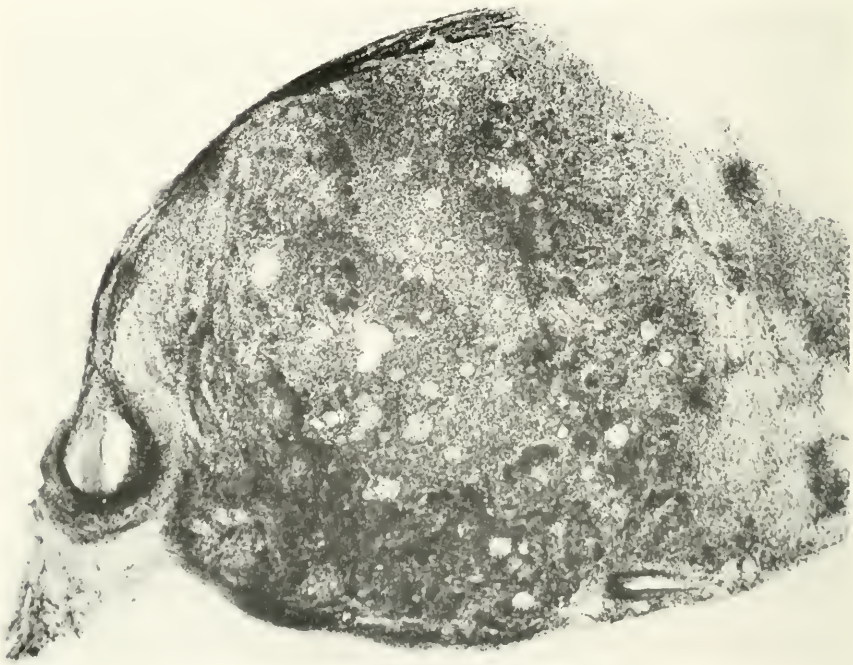
seen frequently in the right eye. The central vision was almost perfect—not quite so good as that of the left. Examining the fundus I found in the upper nasal quadrant, reaching to a point near the upper inner margin of the disc, a large, bluish-gray, rounded, elevated mass, the highest part of which could be seen with +12 D. The projection of the growth was, therefore, about 10 D. into the vitreous, indicating a thickness of a little more than 3 mm. The extent of the growth was about six or eight times that of the optic disc. The vision of the left eye was perfect.

An examination of the field of vision taken March 8, 1915, showed a large defect, including the blind spot, and reaching downward to a meridian 30 degrees below the horizontal, and upward to a meridian almost 60 degrees from the horizontal. The outlines of the field were otherwise normal.

On March 10th I advised the patient to consult Dr. de Schweinitz, informing him that I regarded the neoplasm as a sarcoma. I had never seen a choroidal sarcoma in a patient as young as this one, and I did not desire to enucleate without having the diagnosis corroborated.

On March 12th Dr. de Schweinitz wrote as follows: "I am not at all sure, or perhaps I should say not quite sure, that the lesion is a sarcoma. Naturally, the first diagnosis, as you point out, would be leukosarcoma, but I have seen, and doubtless you have, for I think you have studied the case also, one patient with a very similar-looking lesion, under the care of Randolph, with a diagnosis made over and over again of leukosarcoma, which was certainly inaccurate, as years have gone by, and there never has been any development of the condition of affairs. To be sure, it might be a tuberculous process, in which case the condition would be equally unfortunate for the young woman, but I cannot divest myself of the opinion that it may be some other pathologic lesion, a very unusual form of exudation, perhaps, with cyst formation. To put it in other words, if this patient were under my care, I would state with frankness the possibility of other processes being present, and that in these circumstances it would seem to me justifiable, for a time, to keep the case under observation. I do not believe, even if it proves to be a sarcoma, that such a course,

Case 1.—Neoplasm at margin of optic disc.



Case 1.—Neoplasm under low power.

now usually called watchful waiting, is unjustifiable." He suggested a trial with radium.

Dr. Hamburger was consulted, and it was agreed to have a subcutaneous tuberculin test made. She was admitted into the hospital for this purpose; the test was negative. The patient was observed carefully, almost daily, and the impression was that the growth was becoming larger. On March 15th I noted that since last seen there was an increase of detachment between the papilla and the neoplasm. The detachment reached up to the disc. There was a very small hemorrhagic extravasation on the surface. Where there had at first been fine hemorrhagic points, there was now a mass looking like confluence of a number of these points. On March 25th the patient consulted Dr. Theobald, from whom I received the following opinion:

"I examined Miss C. with much interest, and I cannot make anything out of it except an intraocular growth, in all probability a sarcoma. It is, for the most part, so sharply defined that it does not resemble the inflammatory conditions which sometimes lead us into a mistaken diagnosis of tumor. It is true that the tension is rather below than above normal, but this, of course, is sometimes the case with an intraocular growth. It seems to me that the early enucleation is indicated, for *much may* be gained by this, and as the sight of the eye, in any case, is doomed, there is little or nothing to be lost."

On March 25th the hemorrhagic spot has disappeared. The surface could still be seen with +12 D.

Following the suggestion of Dr. de Schweinitz applications of radium were made to the eye. The treatment was made possible through the kindness of Dr. C. F. Burnam, of the Howard A. Kelly Institute. There were several "massive applications." During this period the eye was watched carefully, but it was not evident that it effected any distinct changes. I therefore advised enucleation of the eye, and the operation was performed on April 11, 1915. The eyeball was given to Dr. Standish McCleary, Professor of Pathology at the University of Maryland. It is proper to mention that when a preliminary report was received from Dr. McCleary on April 20th, I arranged to have a Wassermann test made; for various reasons this had been

regarded as unnecessary before the operation. The result of the test was received a few days later as "doubtful." Dr. Charles E. Simon advised that mercury be given for one week, when a second Wassermann was made which was absolutely negative. On May 5th Dr. McCleary reported:

There is an elliptic mass of tissue on the nasal side of the disc, on the plane of the optic nerve, 0.5 cm. by 0.75 cm., projecting into the interior of the eye. This mass is internal to the choroid, and has pushed the retina away from that tunic, causing a destruction of the pigment layer of the retina over the summit of the growth. Under low magnification the tissue mass is seen to be made up of a number of smaller nodules, each having a distinctly zonal arrangement of its component parts. Two of these nodules are degenerated at the center. The tissue is poorly supplied with blood-vessels. Several giant-cells of the foreign body type can be seen. The picture is typically that of an infectious granuloma, tuberculosis, or syphilis. For examination with higher objectives, sections were also stained to demonstrate tubercle bacillus and treponema pallidum. Most careful search failed to disclose the presence of either organism.

Under high magnification, the nodules consist of lymphocytes, endothelial leukocytes, plasma cells, and a reticulum of fibroblasts and collagen fibrils. A few chromatophores are present in the portion nearest the choroid.

Two of the component nodules of the mass show areas of necrosis, with typical karyorrhexis and caseation. That portion of the growth nearest the retina shows a much more advanced development of the fibrous tissue, and here the pigment-layer of the retina has disappeared. There is an exudate separating the pigmented and neuroepithelial layers of the retina at each extremity of the growth.

This exudate is composed of serum in which are lymphocytes and endothelial leukocytes. The structure and arrangement of the mass are characteristically inflammatory, and nowhere is there the slightest suggestion of true tumor structure. With the von Pirquet and Wassermann tests both negative, and the failure to find either the tubercle bacillus or treponema pallidum in the sections, one is not justified in calling the growth either tuberculous or syphilitic,

yet the structure is such that one of the two is guilty if not proved.

I submitted sections to Dr. Verhoeff, who gave me the following statement, "the tumor is in all probability a solitary tubercle of the choroid. If you will stain a large number of sections differentially, you ought to be able to find some tubercle bacilli in them." (This was done without result.)

Dr. de Schweinitz, to whom I had likewise sent sections, submitted them to Professor Allen Smith, of the University of Pennsylvania, who made the following report, which Dr. de Schweinitz had the kindness to send me:

"Section of eye shows as the important lesion a small mass developed from the choroid close to the nerve. This, which at first glance strongly suggests a diagnosis of sarcoma, is apparently entirely inflammatory. In the section examined there is a central focus of more or less fragmented polynuclear leukocytes, with a developing fibroblastic wall about it. At one point the pus has apparently broken through the latter and a secondary suppurating focus is here developing at the margin of the first. Lateral to these (one on each side in the section) are two fairly defined cellular divisions of the mass, in one of which a smaller but recognizable center of suppuration is forming. All these are enveloped in a mass of round and spindle cells, some of which, near the choroidal base of the growth, are pigmented; some are ordinary young connective-tissue cells; some, from their size and the vesicular type of their nuclei, of endothelial origin; some of the type of small mononuclear leukocytes; and close to the necrotic and suppurative centers there is more or less definite polynuclear leukocytic infiltration. The mass is not well vascularized, but at places angioblasts are evident, and the small choroidal vessels at the base of the growth show distinct enlargement of their endothelial cells. '

"I have hesitated somewhat in concluding as to the 'suppurating' character of the central foci mentioned; but have insufficient basis to conclude otherwise, although the ma-

terial in its staining shows too much chromatin material preserved to be entirely typical. I do not, however, hesitate to distinguish the process as inflammatory rather than neoplastic."

Since the eye was enucleated the patient has been progressing fairly satisfactorily so far as her general health is concerned, though she still complains of frequent and at times severe headaches.

In reviewing the case just described, a number of features should be discussed. The diagnosis of choroidal sarcoma in a patient eighteen years must arouse suspicion. Still, we must bear in mind that Kerschbaumer,* in her work on sarcoma of the eye, in which she reported on 67 cases, found two cases in children and several cases in persons under thirty.†

Syphilitic growths within the eyeball are of very rare occurrence, if we are to judge from the published accounts. I have been able to find only the following references:

According to Baas,‡ true gumma of the choroid had been established in only a single case—in that of Schoebl; and there were no true cases of retinal gummata that had been definitely proved, though there were clinical observations.

Parsons§ says: "True gummata of the choroid characterized by necrosis are of extreme rarity. . . . Only gummatous infiltration has hitherto been observed."

Groenouw‖ states that gummata of the choroid and the retina are very rare; they have the appearance of prominent white masses which arise in the choroid and invade the retina secondarily; they are usually accompanied by iritis, optic

* Das Sarkom des Auges, Wiesbaden, 1900.

† The three cases under twenty years of age are the following: Case 12, a child not over one year of age with epibulbar melanosarcoma; case 27, a male aged three, with angiosarcoma of choroid, and case 20, a girl of sixteen with spindle-cell sarcoma arising in the ciliary body and invading the choroid.

‡ Graefe's Arch., 1898, lxv, p. 3.

§ Pathology of the Eye, London, 1905, ii, 462.

‖ Graefe-Saemisch Handbuch, second ed., xi, Part I, 778.

neuritis, retinal hemorrhages and bulging of the sclera.* It is evident that this picture is quite different from that described in the case reported.

The possibility that the growth was a conglomerate tubercle must also be considered. This condition is likewise exceedingly rare. It is commonly associated with other manifestations of tuberculosis. Such tumors have been generally described as whitish or yellowish white in appearance; in a few instances this has led to the enucleation of the eyeball under the diagnosis of glioma.† In the neighborhood of the tubercular neoplasm there have usually been small miliary deposits. Finally these tumors are ordinarily complicated by signs of inflammation of the conjunctiva, cornea, or iris. It is true that a few cases have been recorded in which there has been complete absence of

* Haas: Zuzammenstellung der . . . Faelle von primaren Erkrankungen des Uvealtraktus. Dissertation, 1880.

Lawson: Trans. Oph. Soc. United Kingdom, vol. iii, p. 117. Large elevated mass at margin of disc containing cholesterin crystals, with extensive choroidoretinitis. The case was probably one of the group of retinitis with massive exudate.

Badal: Arch. d'Opht., 1886, p. 104, 301.

Alexander: Syphilis und Auge, Wiesbaden, 1889, p. 77. A male, aged forty-two, had pronounced luetic symptoms; the right eye was badly affected with vitreous opacities; after these had cleared up, two elevations could be seen; they were evidently very small, not reaching more than 1 mm. or 1½ mm. into the interior. They disappeared under continued antisyphilitic treatment.

Lewin: Arch. f. Derm. und Syph., 1891, p. 1000.

Hirschberg: Lewin Festschrift, 1895, republished in his "Ausgewählte Abhandl., Berlin, 1915, p. 134. He describes two cases; both were severe forms of lues. In both there were large, prominent, rapidly enlarging choroidal masses, producing intense vitreous opacities. The optic nerve was very congested.

Gutmann: Münch. med. Woch., 1902, p. 2098. He found an oval neoplasm near the macula, with hemorrhages in the retina round about, and a paracentral scotoma.

Alt: Ophth. Record, 1908, p. 300 (probably cured cases).

Hanssen: Klin. Monatsbl. f. Augenh., vol. lvi, p. 66 (1916); pathol. report, reviewed in Ophth. Year Book, 1916, p. 140.

† Spalding removed an eye because of supposed glioma, and found conglomerate tubercle on pathologic examination.

Natanson reports two cases of conglomerate tubercle in early childhood in one of which the diagnosis was glioma. See Klinisch. Monatsbl., vol. xlviii, 1, p. 113 (1910). Salzmann, in 1911, reported a similar error in diagnosis, Klinisch. Monatsbl., vol. xlix, II, p. 113.

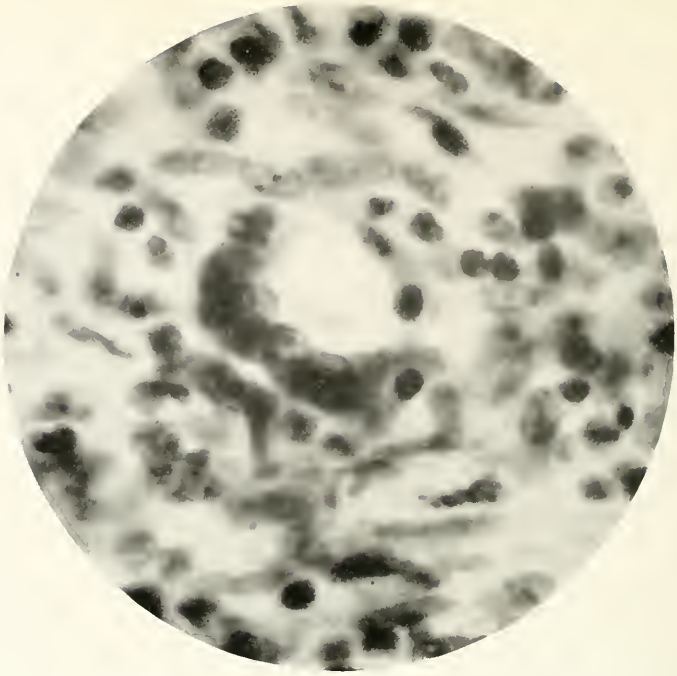
such complication, as in the cases noted by Natanson, Rogman, Solomon, Emanuel and Dupin-Dutemps, and his own (p. 123). Parsons suggests that a solitary tubercle of the choroid may also simulate a choroidal sarcoma. It is evident that there are cases in which the correct diagnosis can be made only by pathologic examination.*

We cannot exclude tubercular origin of the growth, although tubercle bacilli have not been found in spite of the most careful examinations by competent pathologists. In view of the foregoing, I believe that we are justified only in describing the case as a *granuloma of the choroid* of obscure origin. Such a case was described by Frank G. Thomas and George Coats† under the title "Peculiar Granuloma of the Retina"; the case showed a large retinal growth with marked optic neuritis; the trouble followed upon an attack of influenza. The growth was examined pathologically and found to be a large granuloma coming from the retina.

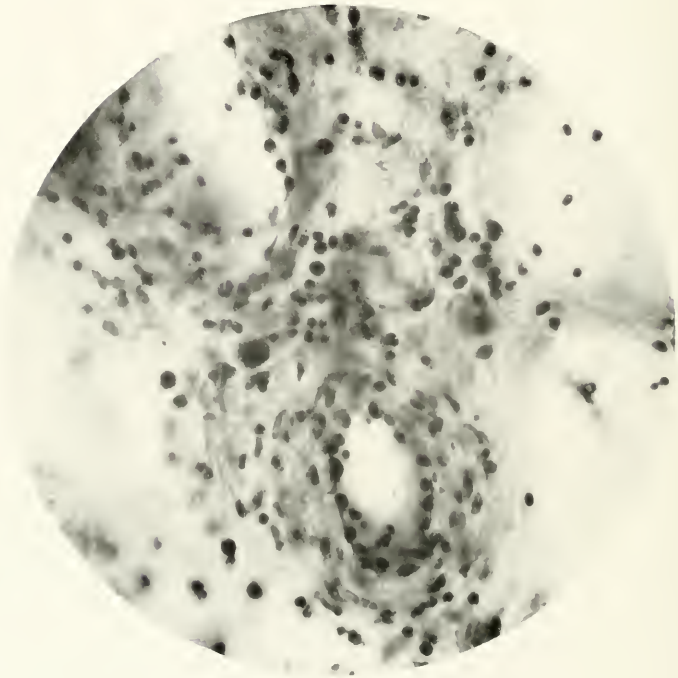
CASE 2.—Charles F., aged twelve years, was first seen on November 16, 1917. The sight of the right eye had been lost, but no definite history could be elicited. The fundus showed intense dilatation of the arteries and veins, both of which were exceedingly tortuous, especially in the temporal half of the retina. In the temporal periphery there was a large neoplasm, rounded, projecting far into the vitreous (its surface could be seen with +17 D.); it was pinkish in appearance, and on its surface large convoluted vessels were conspicuous. The optic disc was blurred in outline and presented the picture of a moderate neuritis. There were numerous small white effusions to the nasal side of the disc, somewhat larger ones arranged in rows, studding the entire macular region. There were also small white exudates in

* For discussion of conglomerate tubercle of choroid see Groenouw in Graefe-Saemisch Handbuch, second ed., vol. xi, part 1, p. 701; Parsons, Pathology of the Eye, vol. II, p. 466 (1905), and Lubarsch-Ostertag's review of years 1910–1913, supplementary volume.

† Transaction of the Ophthalmological Society of the United Kingdom, vol. xxxi, p. 149.



Case 1.—Part of neoplasm under high power, with giant-cell in center.



Case 2.—Organized inflammatory exudate.

the lower portion of the retina. In the lower periphery one large blood-vessel was converted entirely into a white branching streak, accompanied by the white spots just described. In the lower outer field there was a small area of flat detachment. Transillumination, contrary to my expectation, gave no shadow. The ophthalmoscopic picture was very difficult to interpret. The growth had all the appearance of a choroidal sarcoma, but the presence of the large masses of blood-vessels on its surface, and especially the exceedingly high degree of dilatation of the retinal blood-vessels (which were increased in breadth to four and five times the caliber of the vessels in the fellow-eye), were very anomalous. A careful clinical examination at the hands of Dr. Louis P. Hamburger showed that the boy was otherwise quite healthy. Dr. Sydney R. Miller examined the blood and urine with negative results; a Wassermann test and a von Pirquet test were both likewise negative. These results excluded a luetic affection or a solitary tubercle. The condition of marked dilatation of the blood-vessels and the presence of evident disease in the retinal blood-vessels, together with the scattered white exudates in the retina, suggested the possibility that the case belonged to the class first described by Coats under the title "Forms of Retinal Disease with Massive Exudation." In cases of this kind (which I described in a paper read before this Society in 1914) I was much impressed by the prominent, tumor-like masses which they at times present, as well as by the marked disease of the retinal vessels.

The patient was seen by several colleagues, who concurred in the probable malignant nature of the neoplasm and who agreed in the advice that the eye should be enucleated. This operation was performed December 6th, at the Woman's Hospital. The eyeball was placed in the hands of Dr. Standish McCleary for pathologic investigation. His report is as follows:

After formaldehyd fixation a section through the equator of the eye shows a mass 0.9 cm. by 0.6 cm. projecting into the posterior chamber.

Under low-power magnification this mass is very vascular, and the vessels, both in size and number, are excessive. A

recent hemorrhage covers the surface of the mass at one point. There is also diffuse hemorrhage.

Higher magnification shows that the vessels are in various stages of development, some forming and others fully developed. Several of the smaller ones contain hyaline thrombi. The tissue is very cellular, and consists chiefly of what is termed "round-cell exudate," comprising lymphocytes, a few plasma cells, polynuclear leukocytes, and large mononuclear cells. Scattered among this cell complex are numerous fibroblasts and collagen fibrils. The organization of the exudate is more advanced in other places, showing strands of fibrous tissue. Between the pigmented epithelial layer of the retina and the choroid, for a distance equal to one-half the length of the mass, there is a serous exudate containing a few leukocytes, lymphocytes, and pigmented cells.

There is nothing in any part of the mass to suggest a neoplasm, and the picture is typically that of an inflammatory exudate undergoing organization.

Dr. McCleary regarded the case as one of "Retinitis with Massive Exudation."

The two cases described in this paper indicate the difficulty, or even the impossibility, of making a definite diagnosis in some cases of neoplasm of the fundus solely on the ophthalmoscopic appearances.

DISCUSSION.

DR. HIRAM WOODS, Baltimore, Md.: I want to say just a word concerning Dr. Friedenwald's concluding sentence, about the difficulty in making a diagnosis on the ophthalmoscopic appearances. He has been kind enough to let me observe some of these cases with him. I could not make differential diagnosis on the ophthalmoscopic appearance, but the question of transillumination comes up. I recall the generally accepted principle that you get transillumination in non-malignant growths and do not get it in inflammatory processes. Fridenberg, I think, was the first who told us about it. Basing my procedure on that opinion, I enucleated, two or three years ago, the eye of a child three

years old with a large mass in the vitreous, without any evidence whatever of previous inflammatory trouble. The transillumination was completely shut off, and yet it proved to be only an inflammatory deposit. I saw one of the cases that Dr. Friedenwald formerly reported, in which this massive exudate was evidently becoming larger, with inflammatory reaction. I do not believe that we know much of the pathology or what is at the basis of this class of cases.

DR. ARNOLD KNAPP, New York City: One of the cases that Dr. Friedenwald describes (conglomerate subretinal tubercle), I think I have seen two examples of, and in both there was a circumscribed mass characterized by having a curious, dead-white, grayish color. There were no inflammatory symptoms so far as the optic nerve and retina were concerned. Both were in patients around thirty years of age. An unusual feature present in both were some hemorrhages and white spots in the neighborhood of the swelling, degenerative changes, such as are present in certain types of retinal disease. I should like to ask Dr. Friedenwald whether these were observed in his case.

DR. FRIEDENWALD: There was no sign of inflammation or hemorrhage; the retina and optic nerve in the first case were normal. Central vision was almost perfect at the time of enucleation.

DR. KNAPP: Both of the cases are being watched. The diagnosis of a probable solitary tubercle was made and enucleation was not advised. The process has remained stationary in one case for three years, and the other I have not seen for a year.

DR. HARRY FRIEDENWALD: How far did they project?

DR. KNAPP: In the first case six diopters, and in the second eight diopters.

REPORT OF A CASE OF TRANSIENT LEFT HOMONYMOUS HEMIANOPSIA.

HENRY H. TYSON, M.D.,
New York.

On March 27, 1911, M. C., aged twenty-eight years, banker, was referred to me by his physician on account of blurred vision on his left side. He gave a history of awakening on the morning of March 24th, after a champagne spree, with a severe headache, located on the right side of his head, extending from the occiput to and around his right eye. The pain in his head remained until the next day, when he noticed that his vision was indistinct on his left side, and had remained so until the date of his examination. For the relief of his headache he had taken, by order of his physician, acetanilid, 8 grains, and novaspirin, 40 grains, during the twenty-four hours. During the forty-eight hours preceding the loss of vision, according to his statement, he had imbibed four quarts of champagne and numerous alcoholic beverages, viz., silver fizzes, cocktails, etc. He had been accustomed to smoking from 30 to 40 cigarettes daily, and gave an early history of occasional attacks of migraine, commencing when he was eight years of age, and at times numbness of his hands would accompany them. They ceased at the age of twenty-three years. During 1903 Dr. R. F. Weir removed a tumor from his thigh, and the pathologist pronounced it a traumatic sarcoma. Syphilis negative —had gonorrhea during 1905. Tuberculosis, teeth, and sinuses negative. Examination of eyes, March 27, 1911: V. R. E. = 20/15; L. E. = 20/15, with correction sph. +0.25 ⊖ cyl. +0.25 axis 90° = 20/10 both. Muscles, orthophoria. Tension normal. Sensibility of corneæ normal. Pupils 2.5 mm. each; reaction to light, accommodation, and consensually normal. Fields of vision show incomplete left homonymous hemianopsia, with form field contracted to

within eight degrees of fixation on horizontal meridian (Fig. 1). The color-fields were contracted to fixation in similar fields. Eye backgrounds normal, except that the retinal veins were a trifle darker than normal. Pulse, 76; blood-pressure, 138. Blood examination by Dr. Sondern: R. B. C., 5,030,000; W. B. C., 9000; hemoglobin, 68 per cent.; differential count, relative lymphocytosis. Urinalysis: sp. gr. 1015; acidosis, low urea, no albumin, no sugar, no excess indican (faulty metabolism). No symptoms indicating any other nerve lesion.

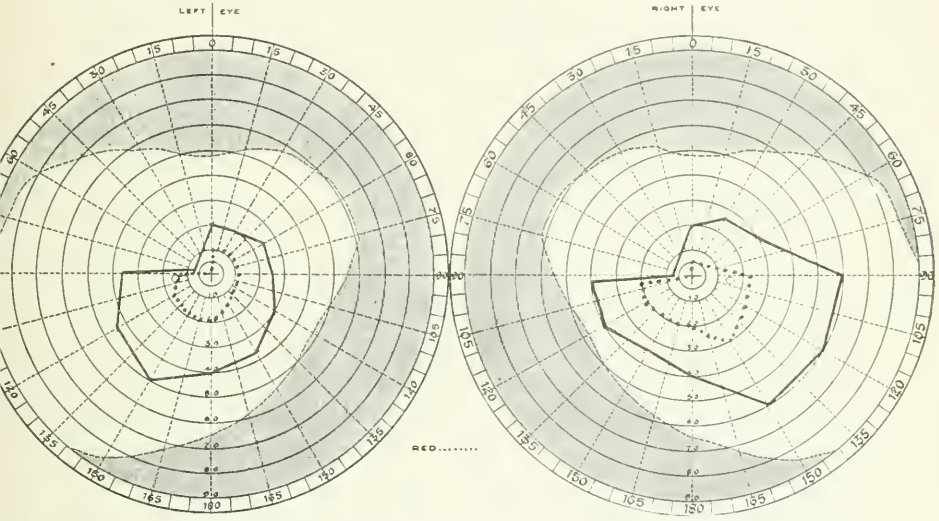


Fig. 1.—Fields, March, 1911. Heavy line, field for white.

Eliminatives and light diet, with avoidance of alcohol and tobacco, were ordered, and nine days later color and form fields were quite normal. On December 7, 1911, he called again for examination. V. R. E. = 20/20; L. E. = 20/20; stated that ten days previously he had had a "bilious headache" after another spree, and that his vision was again blurred, but had not cleared up like the attack in March. He had been drinking large quantities of champagne and other alcoholic beverages during the past month, and had also been smoking his usual number of cigarettes—about

40 daily. Examination of visual fields showed left homonymous superior quadrant anopsia incomplete, the apex of the angle extending to within five degrees of fixation for form, while that for colors was contracted to fixation. The right fields were contracted about 25 degrees (Fig. 2). Eye backgrounds—discs, slight pallor, with edema; retinal veins dilated and tortuous and arteries slightly contracted. On December 20th his form-fields had widened to within ten degrees of normal, while his color-fields remained contracted in left superior quadrants to about fixation, con-

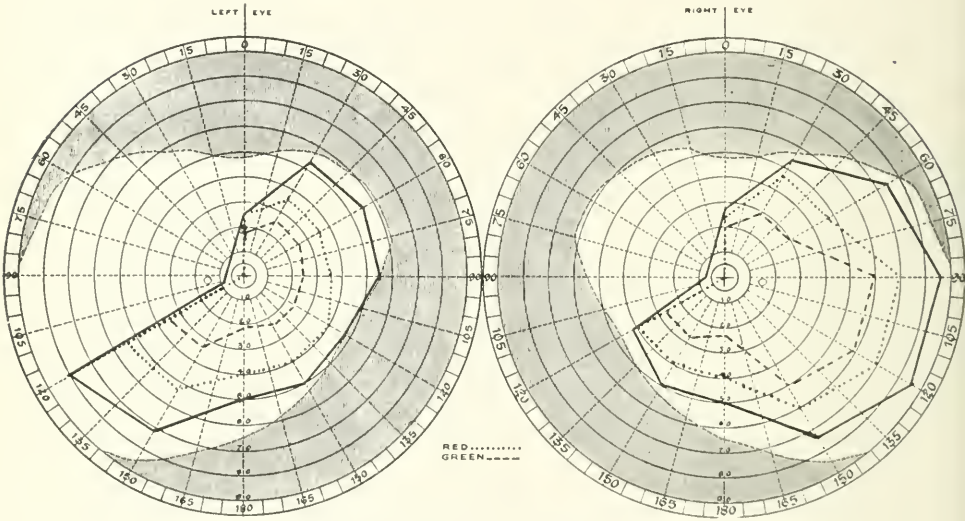


Fig. 2.—Fields, December, 1911. Heavy line, form field.

stituting a left homonymous superior quadrant achromatopsia.

Transient hemianopsia may be found associated with cerebral lues,* uremia, lead poisoning, general paralysis, chronic nicotine poisoning, and in ocular migraine. After careful consideration of the case just reported, I am of the opinion that it belongs in the class of ocular migraines, and

* Oppenheim: Lehrbuch des Nerven-Krankheiten, 1913, vol. ii, p. 1274.

that the previous history of a neoplasm, or the drugs prescribed by his physician for his headache, had no influence as causative factors in producing the hemianopsia. I think that the alcohol and the nicotine, especially the former, were the exciting elements, along with a constitutional migrainous dyscrasia in originating the attacks.

Among others, Peter* reported a case of left homonymous hemianopsia associated with migraine, and Posey† a case with fields somewhat similar to mine, excepting that it was right homonymous hemianopsia and was permanent. As to the localization of the site of the lesion in these cases, we are indebted to Beevor and Collins‡ for their careful study and report of their case of permanent left superior quadrant anopsia, with clinical, microscopic and autopsy findings, the visual fields in which were very similar to those in case under consideration. "They found at autopsy an occlusive lesion of the right posterior calcarine artery, which caused destruction of the inferior third of the cuneus, including all the cortex above and below the calcarine fissure, with the exception of a small point anteriorly. Careful microscopic study showed that the lesion was limited entirely to the cortex. They concluded that the lower third, including the upper and lower lips of the calcarine fissure, represented the upper visual quadrants, while the upper two-thirds of the cuneus represented the inferior quadrants." In order to account for the production of the transient anopsias in the left superior quadrants in migraine, we would be obliged to postulate a prolonged spasm of the right posterior calcarine artery, with a consequent ischemia and possible edema (angioneurotic edema), with subsequent disappearance of same. The extent of organic changes, if any follow, would depend upon the toxicity of the exciting

* Peter: Principles and Practice of Perimetry, p. 203.

† Posey: Journal American Medical Association, May 8, 1915.

‡ Beevor and Collins: Brain, 1904, p. 153.

cause, and the length of time the tissues were deprived of their nutrition on account of the disturbed circulation.

The interesting features of the case are: 1. The transitory nature of the first attack, which persisted nine days, with restoration of visual fields, and the recurrence eight months later with recovery in form field but permanent left homonymous superior quadrant achromatopsia remaining.

2. The fact that alcohol and tobacco were probably the exciting cause of the cerebral angioneurosis.

DISCUSSION.

Dr. J. H. Claiborne, New York: Dr. Tyson has referred to a case I reported several years ago before this Society. The patient was a man of middle age who was accustomed to getting on periodic sprees. Being quite ill after one of these, he took a very large dose of salicylate of soda. He had a sudden disturbance of vision, the nature of which could not be discovered by his consultant. He had a homonymous tetranopsia which persisted. I have seen him in the last year or more, and it is still present. Apropos of this case, though not exactly similar, I would like to report the case of a young man of sixteen with a high degree of compound hyperopic astigmatism in the right eye and less in the left. A week before I saw him he was taken suddenly blind, as he thought, but on questioning him he stated the right side of the objects had disappeared. At the same time the right arm and entire right side were numb. The optic nerves were normal, and hemianopsia had disappeared when I saw him. There was no difference in sensation or in muscular power on the two sides of the body, and there were no areas of anesthesia. I have never seen a case of this kind of transient fugacious hemianopsia in a person so young. He did not appear to be a neurotic subject, but I was inclined to think he was after a searching cross-examination. In my opinion high degrees of refractive error do not produce these symptoms. I think there is an underlying cause, while the refractive error may precipitate the attacks.

Dr. Wilder: Were there any areas of anesthesia?

Dr. Claiborne: Absolutely none.

A PRACTICAL POINT IN THE TECHNIC OF CORNEAL TATTOOING, THE VALUE OF WHICH IS NOT COMMONLY RECOGNIZED.

SAMUEL THEOBALD, M.D.,
Baltimore, Md.

A case of rather extensive and very irregularly outlined corneal opacity, the result of a penetrating wound of the cornea with laceration of the iris in early childhood, which recently came into my hands for tattooing, afforded some ground for cogitation when the point was reached of deciding how I should proceed in the matter. A minimum of traumatism, because of the grave nature of the injury which the eye had suffered, and the insertion of the India-ink just where it was needed, and *only there*, were the desiderata.

At the first sitting, following my usual practice, I applied the ink to the cornea and pricked it in with a Weiss cataract needle. It was almost at once manifest, however, that this procedure was ill adapted to meet the conditions, for the ink completely obscured the field of operation, and it was quite impossible to distinguish between the clear cornea and the irregularly outlined corneal opacity; furthermore, I could feel no assurance that I might not be stabbing, more than once, the same spot in the leukomatous tissue.

A trivial amount of irritation followed the operation, and in two days the condition of the eye warranted a repetition of the tattooing. This time, however, I reversed the order of procedure, first using the needle and afterward rubbing in the ink. With an unobscured field I could see distinctly the outlines of the corneal opacity, and tell exactly where the little oblique stabs should be made, and, besides, it was

easy to avoid making more than one stab at the same spot. The ink was applied with a small cotton mop and gently rubbed in with a Daviel curet. The cosmetic effect, as observed the next day, was most satisfactory, and only one more sitting, after the lapse of another twenty-four hours,—the same procedure being then followed,—was required to complete what I may, perhaps, be pardoned for calling the requisite "camouflage."

A cursory examination of the literature bearing upon the subject in my library showed that the procedure which I had employed with so much satisfaction was not novel; but, on the other hand, it convinced me that its advantages were little known, for I found that a large majority of the authorities, in describing the operation of corneal tattooing, spoke only of the application of the ink to the cornea before the use of the needle or of pricking the ink into the cornea by means of a grooved needle. And it is for this reason that I have thought it worth while to make this communication.

Cocain in 4 per cent. solution, supplemented by a single application of a 1:1000 adrenalin solution, afforded very satisfactory anesthesia. Murdoch's speculum was used to keep the lids apart, but fixation forceps were not employed, the eye being steadied by the finger-tip. The India-ink, which there is good warrant for believing contains no micro-organisms, was not sterilized, but was rubbed up with a few drops of sterile water upon a suitable sterilized glass dish.

HYPERMETROPIA RESPONSIBLE FOR HETEROPHORIA, ASTIGMATISM, AND MYOPIA.

S. M. PAYNE, M.D.,
New York.

In the year 1888 the Stevens theories and claims of cures of certain ailments by partial tenotomies caused so much interest that a committee of ophthalmologists and neurologists was appointed to investigate them. In the writer's belief, the results of the new treatment were not satisfactory, but it was adopted by a large number of ophthalmologists all over the country. They were divided into the following six groups as to cause of deviation: muscles too strong, too weak, too long, too short, attached too near the cornea, and malformation of the orbit. Stevens, in his book on Functional Nervous Diseases, states that the superior rectus of one eye is too strong, or the inferior rectus of its fellow is too strong. In either case he calls the vertical tendency "hyperphoria," and cuts the superior rectus of the eye that is on the higher plane. If that fails to correct the tendency, he cuts the inferior rectus of the fellow-eye.

During the investigation by the committee the writer had the good fortune to measure the tendency before and after these operations. During the first period of the operations the writer discovered that the eye operated on was the fixing eye, and, of course, was the eye on the horizontal plane, while its fellow was below it. A common-sense view afforded a simple explanation. Instinctively, man or animal fixes with the better eye, whether or not the fellow-eye has a tendency to deviate. The writer saw cases of tenotomy of superior rectus in eyes with vision 20/20 to 20/15, while the

vision of their fellows was 20/30 or 20/40 or even less, this faulty vision being due to a greater error of refraction. Thoroughly convinced that the vertical tendency to deviate was due to the difference in the error of refraction, and that the tendency was downward, the writer worked out the causes and discovered that the fault was not in the muscles, but in error of refraction. Those muscles act instinctively to aid the fixing eye by a greater muscular effort, just as an eye with distorting vision produced by lesion causes the eye instinctively to turn. If the fixing eye is better than the two combined, the affected one is discarded.

On this subject a paper was written and read before the ophthalmic section of the Academy of Medicine in 1893, with a report of 200 cases showing the following:

Refraction the same:			
Esophoria	70		(90.1 per cent.)
Exophoria	21		
	—	91	
Hypophoria	1		
Hypoesophoria	5		(9.9 per cent.)
Hypoexophoria	4		
	—	10	
		101	
Refraction different:			
Horizontal tendency	19		(19.2 per cent.)
Vertical tendency	80		(80.8 per cent.)
	99		
Tendency downward	65		(81.25 per cent.)
Tendency upward	15		(18.75 per cent.)
	80		

TORSION.

After reading Dr. Savage's paper on Insufficiency of the Oblique Muscles, the writer began to look for cases showing those defects, and found only three, all of which were in eyes having a different error of refraction. This led the writer to prepare and read a second paper before the ophthalmic

section in 1898. In this collection of 514 cases the following conditions appeared:

Refraction the same:		
Horizontal tendency	194	(81 per cent.)
Vertical tendency	45	(18 per cent.)
	239	
Refraction different:		
Horizontal tendency	42	(14.55 per cent.)
Vertical tendency	233	(85.45 per cent.)
	275	
Hyperphoria	74	(31+ per cent.)
Hypophoria	159	(68+ per cent.)
	233	

Of these two papers combined, 340 showed the same error of refraction, the tendency to deviate being confined to the horizontal plane, 83.82 per cent., and those showing a vertical tendency, 16.18 per cent.

The remaining 374 cases were anisometropic, showing a horizontal tendency to deviate, of 16.32 per cent., and a vertical tendency of 83.68 per cent.

It thus appears that the totals in the two classes of cases are virtually reversed, the percentage being about 84 and 16 per cent. respectively in each class, the difference being about one-seventh of 1 per cent.

The third paper on this subject was published in 1904, in the Archives of Ophthalmology, vol. xxxiii, with a report of only a dozen cases.

EMMETROPIA.

The writer believes that all the external ocular muscles supplied by the third nerve act with the ciliary muscle in accommodation. The four recti muscles are so attached to the globe of the eye that, acting singly, they turn in the direction of that muscle. Any two muscles acting together turn the eyeball in the diagonal of these directions, as in looking around a room. The oblique muscles rotate the

eyeball on its anteroposterior axis, keeping the eye in its vertical plane.

The internus and inferior rectus are governed by a common tendon,—the ligament of Zinn,—which is attached around the circumference of the optic foramen except at its upper and outer part. These two muscles are larger and stronger than any other two muscles combined, the internus being the larger and stronger. In accommodation they act together, of course, downward and inward. The superior recti are the thinnest and narrowest of the four recti muscles which pull the eye upward and inward. The inferior obliques, thin, narrow muscles, pull the eye upward and outward, and both are supplied by the third nerve. If the inferior oblique turns the eye outward above to keep it on its vertical plane in accommodation, what action could the superior rectus take except to relax? It is hardly logical to assume that it is the only idle muscle in accommodation supplied by the third nerve. It must be included in the process of accommodation, the superior rectus and inferior obliques counteracting or counterbalancing each other, and holding the upper part of the eye in its original position, while the internus and inferior rectus turn it inward and downward. At the same time the externus and superior oblique relax, so as to place the lower part of the eye directly under the upper part, thus keeping the eye in its vertical plane, turning downward and inward.

When hypermetropia is the same in both eyes, and the vision is normal, the tendency to converge is equal in the two eyes. If esotropia develops, one eye turns downward and inward by increased action of the two muscles to avoid diplopia. We should call this a voluntary contraction, because it is independent of the amount of stress necessary to work with the fellow-eye. That this is independent of the action necessary for accommodation is shown by the fact that there is usually no latent hypermetropia. That is, the

eye would accept a +4 D., while the fixing eye would probably have a manifest of not more than 1 D. When under atropin they would both accept +4 D. When they work together (absence of squint), the manifest varies from nothing to probably the full amount of error. When the hypermetropia is different, we have either hypophoria or hypoesophoria, more often the latter. The hypophoria is in the more hypermetropic eye, while the fixing eye is on the horizontal plane.

When hypermetropia differs in the two eyes, and each accommodates to correct its error, the eye with the greater error turns more inward, as it turns more downward and may be thrown on a different plane. Torsion is more apt to be found in myopic eyes differing in refraction. As they cannot work together, the discarded eye is turned outward and downward by the contraction of the externus and superior oblique.

Pseudo- and True Heterophoria.

Dr. Savage claims that pseudo-heterophoria is the amount that glasses can correct; that true heterophoria is the uncorrected esophoria in hypermetropia, the uncorrected exophoria in myopia, exophoria in hypermetropia, and esophoria in myopia. If it can be shown that what Dr. Savage calls "true" is pseudo-heterophoria, something worth while has been gained. Our literature on the subject of accommodation is with emmetropic eyes or eyes made emmetropic by correcting glasses. This is preliminary to what we wish to prove. We will take 13 inches as a comfortable distance for an emmetropic eye. The first impulse is to see distinctly, causing the crystalline lens to increase 3 D. The second impulse is to avoid diplopia by converging to a point at 13 inches. So long as nature provides an emmetropic eye to go with accommodation and convergence to a given point, all goes well. But when eyes hyperopic 3 D. have to

accommodate 6 D., an attempt to converge to a point 13 inches away, when the natural convergence would be six and a half inches, all goes wrong. This convergence to 6½ inches is prevented by a volunteer contraction of the externi and superior obliques—the counteraction muscles; unless it is possible for the interni and inferior recti to contract, only half the amount of the ciliary muscle. In such cases, if we do not get a convergent squint, we have either esophoria or exophoria. The cases of exophoria in hypermetropia can be attributed to two causes, working together or separately:

1. The strain or fatigue of the ciliary muscle may come first, which would be indicated by a blurring or, as patients often say, everything turns black before their eyes. This would be followed by a relaxation of the internal and inferior recti muscles.

2. The fatigue in convergence is more likely to come first, as they are antagonized by two opposing sets of muscles, the externi and the superior obliques, to offset the extra physiologic action of the interni in accommodation to a given point, indicated by the extra increased curvature of the crystalline lenses. The indication of the interni fatigue is either a separation of the letters into two sets of letters, or a tendency to do so, indicated by the broadening out of each letter, making the letters unrecognizable. This condition is described by the patient as "the letters dancing all over the page." This may mean either exophoria or esophoria, or alternating struggles against the ciliary muscles to hold convergence at a distance of 3 D. We may have exophoria for the near and esophoria for the distance, which is not at all uncommon, and in other cases exophoria for both far and near, which is often found.

When the hypermetropia is different, the eye with the greater error moves inward and downward, on account of greater muscular effort to correct the difference. Thus hypoesophoria 80 per cent. The hyperesophoria of 20 per cent. is

accounted for by the internus and inferior rectus becoming tired, for the same reason that we have exophoria in hypermetropic eyes the same.

If the full correction for the hypermetropia is made, Dr. Savage's true heterophoria turns out to be the pseudo-heterophoria. When this full correction is given, the interni and ciliary muscles act in harmony; and neither the exophoria or hypoexophoria any longer exists either near or at the distance. Recently, considerable attention has been given to ciliary muscle fatigue by some of our distinguished ophthalmologists. The most recent was by the Chairman of a Committee of the ophthalmic section of the American Medical Association for eye muscle research. This report on the ciliary muscle gave the measurement of fatigue at different ages.

While such reports are interesting, their practical value is questionable. If all errors of refraction are corrected, ciliary muscle fatigue will be confined practically to emmetropic presbyopic eyes, and to those of younger people who hold their work too near the eyes. It is the opinion of some ophthalmologists that amblyopia, in hypermetropia, precedes the squint, and of others that amblyopia comes subsequent to the squint from the non-use of the eye. As this paper is confined strictly to muscles and refraction, no other amblyopic cases are considered.

In the writer's opinion, excessive accommodation causes double vision, followed by alternating squint, which later on becomes a permanent squint of one eye. While the writer agrees with the latter group that the amblyopia is subsequent to the squint, he does not believe that it is directly due to the squint. We have all seen eyes where the squint had existed for years with normal vision in the squinting eye, with or without a glass. The reason for this is that the accommodation in that eye has continued, and all rays of light have fallen upon the retina, including the macula, with the

same sharp focus as that of the fellow-eye. This continued sharp focus keeps the retina active, notwithstanding images are ignored or suppressed. When we find a squint of long standing with amblyopia, we find the hypermetropia to be 4 D. or over, with absolutely no accommodation. This produces the amblyopia, because the rays of light fall upon the retina and macula under circles of diffusion. With no focus to stimulate the retina, the eye becomes amblyopic. This eye will show under the test the total error, while its fellow will show about one-fourth. If the two eyes are placed under atropin, we shall probably find that the fixing eye has the same amount of error, with no increase in the refraction of the squinting eye. We have also noticed that very high degrees of hypermetropia, such as 6 or 7 D., do not have squint. Such cases we shall find are amblyopic unless glasses have been worn to correct the error.

Esophoria in Myopia.

Esophoria in myopia is not very common and is found when myopia is the same in both eyes. In these cases we have no accommodation or less than the amount required to stimulate convergence to the point of fixation. The interni take on an independent contraction to produce sufficient convergence to prevent crossed diplopia. A forced action of an individual muscle outside the laws of accommodation and associated movements of the eyes may produce heterophoria, and in the case of myopia produce esophoria. The myopia corrected will force the eyes to accommodate, as in emmetropia, and the convergence produced by the action of the ciliary muscle will correct the esophoria. When the eyes cease to maintain binocular single vision, for the want of a stimulating influence of the ciliary muscle, one eye begets a tendency to diverge and soon becomes more near-sighted. It then begins to turn outward and downward. When the prism test is made to determine the amount of deviation,

the eye will move further outward or downward to avoid fusing with the other light. When the externus and superior oblique can no longer overcome the prisms, they give up, and the larger light crosses to the other side and may move above or below and back again to the right or left. It seems that any of the six muscles are liable to come into play to avoid fusing. When these muscles become exhausted in their efforts and the lights are finally brought together, the eyes see a smaller and a more defined light in the larger and more blurred light. Unless the vision is very faulty under the correction, the eyes will work together when the image is made the same size on the retina, even in cases of actual divergence, and even after partial tenotomies, when the muscles are not displaced by the cutting.

While the cases above reported show about 84 per cent. horizontal tendency, when the refraction is the same, and about 84 per cent. vertical tendency in anisometropia, about 20 per cent. of the 84 per cent. are hyperphoria and 80 per cent. hypophoria. The cases where refraction is the same show 20 per cent. exophoria in hypermetropia and about 20 per cent. esophoria in myopia; so the exceptions to the rule are about the same.

REGULAR ASTIGMATISM.

During the year 1892 the writer adopted the theory that the greater curvature of the cornea is in the horizontal instead of the vertical axis in more than 50 per cent. of the cases after the age of fifty years.

A paper was subsequently prepared and read at the ophthalmic section of the New York Academy of Medicine in 1894. The following is a summary of the 1071 cases therein referred to, grouping those, according to age, in the horizontal axis. Age ten up, 27 per cent.; twenty up, 29 per cent.; thirty up, 34.4 per cent.; forty up, 47 per cent.; forty-

five up, 49.3 per cent.; fifty up, 54.4 per cent.; sixty up, 64.3 per cent.; seventy up, 100 per cent.

It was observed that oblique axis increased with age below axes 45 and 135, but those cases were not included. No case of astigmatism was found in children under six years of age, and only about a dozen in children under ten. These were excluded.

At the beginning of the writer's studies the text-book stated that astigmatism was either congenital or acquired. When the theory that the axis in the greater curvature of the cornea changed with age was adopted, a cognate idea was presented; it was observed that in the majority of cases the defect was acquired and that the cases of a high degree were congenital and probably the only ones.

Among the thousands of cases examined, from which the 1071 were selected, no cases of congenital origin were discovered. Still the congenital theory was so widely held among eminent specialists that the writer hesitated to publish his convictions. But an added experience of a quarter of a century has dissolved all doubt. Ophthalmologists agree that, as a rule, babies at birth are hypermetropic, and, as they grow, the eyes become more nearly spheric until the majority become round or emmetropic. Add to this the fact that no cases of astigmatism are found under the age of six in the former list of 1071 cases, or the present list of 1752, we are forced to the conclusion that astigmatism begins at the kindergarten age, say from six to ten, due to hypermetropia, at a time when near eye-work is very light.

Assuming that the primary cause of astigmatism is hypermetropia, we proceed to consider the unsettled question, "How is astigmatism produced?" The distinguished writers Fuchs, Juler, Donders, and Schmidt-Rimpler locate the seat of astigmatism in the cornea. Landolt says it is occasionally formed by the crystalline lens. Fuchs, Landolt, and others speak of astigmatism as congenital or acquired, either from

irregular development or an irregular action of the eye. They all state that the greater curvature of the cornea lies closer to the vertical meridian than to the horizontal. When they say it is congenital, there is no more to be said; when they say it is acquired, some of them speak of the lids as the cause. They also speak of an increased curvature of the crystalline lens in the opposite axis to the greater curvature of the cornea, to correct the corneal astigmatism entirely in some cases and in others overcorrect it, as Landolt mentions and claims to have proved by the use of atropin.

That the crystalline lens becomes astigmatic in order to correct the corneal astigmatism is, we believe, universally accepted, but we are not aware that any one believes the crystalline lens produces it when there is no corneal astigmatism. The crystalline astigmatism is only a remedy for the corneal astigmatism.

The purpose of the lids in astigmatic cases is to cut off the end rays on the macula, so as to square the letters and make them more readable. The lids also trombone in myopia and high degrees of hypermetropia in order to cut off a part of the rays to diminish the circles of diffusion. When these three classes of cases are corrected with glasses, the lids cease to trombone and furnish no evidence of having produced astigmatism. If the lids produce permanent astigmatism, this pressure would come directly above and below the cornea, thus making the vertical curvature greater. If this pressure were continuous, we might have some reason for believing that the lids cause astigmatism, partly closing to produce vision, if only for a moment.

The writer has endeavored to eliminate the three universally accepted causes of astigmatism in order to bring forward what he believes to be the true cause, namely, the external ocular muscles. The six are all opposing muscles, and all work together in accommodation. We begin life with hypermetropic eyes. A child's mental capacity at first

is not strong enough to fix intently on any object, so no direct image is formed on the retina. When he gets away from his mother's lap and learns to walk, his mind is still not sufficiently developed to form a sharp focus on objects. This weakness may produce convergent squint, though not astigmatism, as the eyes are not fixed long enough on objects, and then they are constantly changing to distant objects.

When children reach the kindergarten age, as the hours are short and the study is light, only a few cases of astigmatism develop. Only 20 plus and 9 minus were found—a total of 29 of the 1752. During the school years from ten to twenty more concentration is required and longer hours, and astigmatism is increased to 82 plus and 58 minus, total, 140. From twenty to thirty, when a large percentage have entered the commercial world, and some are still in college, the work is harder, with longer hours. The number in this decade is 218 plus and 143 minus, total, 361. In the thirties the work is still more trying, as it is the time entirely of practical application of the knowledge they acquire in the second and third decades. The number is increased to 277 plus and 185 minus, total, 462. In the forties we find a slight decrease, to 230 plus and 146 minus, total, 376. This is accounted for by half of the fifth decade being in the presbyopic age. In this period we find the increased vertical curvature of the cornea decreased 11.6 per cent., while the obliques above axis 45 degrees and 135 degrees, including axis 90 degrees, decreased only 7 per cent. This means either that the axis is shifting from the vertical to the oblique above axis 45 degrees and 135 degrees, then later below and finally to the horizontal, or that the astigmatism disappears first in axis 90 degrees and second above the 45 degrees, and later on in life there is a decrease below axis 45 degrees until the astigmatism is almost entirely confined to axis 180 degrees for plus glass, and 90 degrees for minus glass.

If we are willing to admit that the four external ocular

muscles supplied by the third nerve act in accommodation and the fourth and sixth nerves supplying superior oblique and external muscles counteract at the time of accommodation, then we are obliged to acknowledge that all these muscles act in correcting hypermetropia at the distance.

These muscles are inserted to the sclera near the cornea in such a way that any one muscle may have more or less a countereffect upon any other muscle, unless it should be the interni and inferior recti, which are from the same origin; still, in a way, they oppose each other as they pull downward and inward instead of directly downward or inward. The superior recti guide the inner and upper direction of the eyes to prevent them from being too much under control of the inferior recti. The inferior oblique acts in accommodation to counteract the superior recti and keeps the upper part of the eye in the vertical plane, just as it would be when at rest. The superior oblique relaxes to allow the inferior recti to hold the eye below in its vertical plane, while the eye is turning downward and inward. In this particular action the superior and inferior obliques are allies, because the relaxation of the superior oblique would not be sufficient if the inferior oblique did not prevent the eye from tipping inward above. On the other hand, the upward action of the inferior oblique can prevent too much torsion by the relaxation of the superior oblique. All these actions are to prevent torsion. Besides these, we have the direct opposing actions, the superior oblique against the superior recti and the externi against the interni, which is shown by paralysis of any single muscle, as the eye turns from that muscle in the direction of the opposite muscle.

We must keep in mind that hypermetropia is corrected from the time the eyes are opened in the morning until closed at night by accommodation for the distance, except in high degrees, when the eyes frequently blur to give the muscles a rest. Another exception is when the hypermetropia is so

great that all attempts to correct it are abandoned. These cases become amblyopic. This hypermetropia runs from 5 D. to 7 D., and in some cases more, and vision is usually 20/100 or 20/70.

We have another class of amblyopia: compound hyperopic astigmatism about equally divided between the sphere and cylinder, total, 5, 6, or 7 D. These cases, in my opinion, struggle to correct the error, change half the error into astigmatism, without being able to accommodate to correct the remaining spheric part. This gives a double lined diffusion instead of circle diffusion. (The writer's explanation in his paper, published in 1894, on the changed spheric cornea to spherocylindric by the action of the muscles, will be the same here, as time has not changed his opinion.)

Our experience in taking the tension of an eye with the fingers shows us how easily the curvature of the eyeball is increased between two fingers. Why should not the curvature of the eyeball be increased by two opposing muscles? In fact, the insertion of these muscles is practically where the fingers are placed in taking the tension. If the pulling of the opposing muscles can have a like effect as the fingers, we not only have an increased curvature of the eyeball but of the cornea as well. As the eye approaches the presbyopic age the ciliary muscle has less effect on the eyeball, as it is gradually less soft and less yielding to the opposing muscles. Thus, the accommodation begins to subside and the astigmatic error changes into a spheric error. Many of the writer's private cases have demonstrated this fact.

The writer has collected an additional list of 1221 cases from his office records, which afford further proof that muscles cause astigmatism. In compound hypermetropic astigmatism, when the sphere is the same, we have corresponding axes in the astigmatism. The table shows this to be true in 90.4 per cent. against 9.6 per cent. different axes; also that the axes differ when the spheres differ. These cases show

only 25 per cent. corresponding and 75 per cent. different axes. This proof holds good with both compound myopic and simple astigmatism; also in mixed, when spherocylindric glasses are ordered the same or different.

We sometimes overcorrect the astigmatism at the expense of the spheric or the reverse, as the eye may see the same with a slight overcorrection of the other, but to increase one without diminishing the other reduces the vision. If this be true, the theory advanced may be wholly true. The reason as it looks to the writer is this: The same sphere requires the same accommodation which causes a corresponding axis. Anisometropia, requiring more accommodation in one eye than in the other, causes a torsion of that eye which makes a different axis; or if the accommodation is suspended in that eye, a torsion with axis different ensues on the reverse side.

In other papers the writer has stated that when astigmatism exists, whether the same, different, or only in one eye, the hypermetropia, if any, will be found to be the same, with few exceptions. The usual exceptions occur when there is a decided difference from 1 D. to 4 or 5 D. This is true also with no astigmatism. Only 3 cases were found in 19 that had less than 1 D. difference in simple hypermetropia. With astigmatism only 5 in 17 cases with less than 1 D. spheric difference. Total of 36 cases, 28 more and 8 less than 1 D. difference, 78 per cent. and 22 per cent. The apparent reason for this is that if astigmatism develops in anisometropia, it begins first in the eye of the greater hypermetropia by changing the difference into astigmatism, making the spheric part the same. If astigmatism is brought about by excessive accommodation, it comes first in the eye with the greater error, as it requires more effort to correct this eye by trying to equalize the vision of the two eyes. This fails when the difference is too great. That is why we seldom see the difference less than 1 D. If any further astigmatism develops, it is the same in both eyes, leaving the spheric error still the same.

Among 23 c.c. squint cases, all those found under the age of six were spheric only. The only astigmatic cases were ages ten and eleven. These cases were straightened by prescribing the ophthalmoscopic measurement, and verified with the test-lenses in only the two astigmatic cases. Only in one was atropin used, and that was in the astigmatic case aged eleven. The only other case above the age of the two astigmatic cases was age sixteen, which may have been tested with the lenses; but, as it was not an astigmatic case, + 4 D. was ordered, as found by the ophthalmoscope. The other 20 ages ranged from nine years down to twenty-two months.

In looking over the anisometropic eyes and same spheric error to see what influence each had on the axis of the astigmatism, the same books were examined, and the cases with no astigmatism were copied to get the percentage of same sphere and different. The result follows:

Spherical same 1433 cases	97½ per cent.
Spherical different 38 cases	2½ per cent.
Spherocylinder same 372 cases	94 per cent.
Spherocylinder different 26 cases	6 per cent.

In conclusion the writer restates what he has attempted to prove by the several groups of cases in the present findings of 2974 astigmatic and 1818 spheric cases—4792.

First: Astigmatism is not congenital, not acquired by the crystalline lens, and not by the action of the lids; because none of them would start the greater curvature of the cornea in the vertical axis at 100 per cent. under age ten and gradually change the axis of the greater curvature 100 per cent. in the horizontal axis at the age of seventy. This revolution is complete at the end of the ciliary muscle action. After this age, when the ciliary muscle has ceased to accommodate, the external muscle cannot assist in accommodation, but acts only in convergence and in associated movements.

Second: The axis of astigmatism is largely controlled by the sphere; that is, the axis corresponds when the sphere is the same and differs when the sphere is different. In the

latter case we may find torsion, but not when the sphere is the same. The exception to this rule is small.

CAUSE OF MYOPIA.

It is an accepted fact that hypermetropic eyes become round or nearly round at full growth, and in some cases myopic. In myopia the increase in the anteroposterior diameter is thought to be due to the thinning of the sclera posteriorly. This has been demonstrated by microscopic examination on sections of the sclera, as well as by our ophthalmoscopic findings. The writer believes that this posterior thinning begins in the advanced stages of myopia, due to a tendency to diverge, with a more rapid increase in actual divergence. In low degrees of myopia the fundus usually looks normal, and we seldom see posterior staphyloma in eyes of less than 8 D. The sclera when normal is thicker posteriorly and gradually becomes thinner as it approaches the cornea, so the thinnest part is anterior to the insertion of the six muscles. It is quite probable that the constant correcting of hypermetropia in children is not entirely by the ciliary muscles, but is aided by the six external ocular muscles attached to the sclera at a corresponding distance from the cornea. It seems reasonable to suppose that these muscles just back of the thinnest part of the sclera would depress that part of the sclera including the cornea, producing in a measure conical cornea; that is, a deeper anterior chamber. Hypermetropic eyes that develop into emmetropic at maturity may be due to this muscle action, or at least influenced by it. Children who are obliged to hold their book at 8 inches or less to see the print increase the accommodation 5 D. on probably 4 or 5 D. distant error. This great strain to correct 10 D. may start a low grade of sclerochoroiditis which may be the starting-point of myopia. Those of hereditary tendency are more easily influenced. This thinning of the sclera is occasionally

indicated by a choroidal ring about equal distance from the insertion of the muscles and the cornea.

HYPERMETROPIC ASTIGMATISM. EVOLUTION IN THE GREATER CURVATURE OF THE CORNEA FROM THE VERTICAL TO THE HORIZONTAL AXIS.

AGE	TOTAL	AX.	AX.	AX.	AX.	AX.	AX.	AX.	PER CENT.	PER CENT.	PER CENT.	PER CENT.	PER CENT.	PER CENT.	PER CENT.
		V–VO	V	VO	45 135	H–HO	H	HO	V–VO	V	OV	45 135	H–HO	H	H
6–10	20	20	10	10	0	0	0	0	100	50	50	0	0	0	0
10–20	82	68	38	30	0	14	14	0	83 −	16.34	36.58 +	0	17	17	0
20–30	218	156	77	79	11	51	29	22	71.5	35.3 +	36.2 +	5	23.5	13	10.5
30–40	277	214	120	94	13	50	26	24	77	13.32	33.92	4.6	18	9.3 +	8.6 +
40–50	229	161	73	88	8	60	47	13	70	32.3	38.4	3.4 +	26	20.5 +	5.6 +
50–60	168	75	43	32	7	86	55	31	44.6	25.5 +	19 +	1 +	57.1	30	18.4 +
60–70	67	16	5	11	4	47	31	16	23.8	7.4 +	16.4 +	5.9 +	70	16.2	23.8 +
70 up	38	4	0	4	0	34	30	4	10.5	0	10.5	0	89.4	78.9	10.5

MYOPIC ASTIGMATISM. EVOLUTION IN THE GREATER CURVATURE OF THE CORNEA FROM THE VERTICAL TO THE HORIZONTAL AXIS.

AGE	TOTAL	AX.	AX.	AX.	AX.	AX.	AX.	AX.	PER CENT.	PER CENT.	PER CENT.	PER CENT.			
		V	OV	V–OV	15	OH	H	H–OH	V	OV	V–OV	45	OH	H	H–OH
6–10	9	6	3	9	0	0	0	0	66⅔	33⅓	100	0	0	0	0
10–20	58	17	30	47	1	8	2	10	29.3	51.7 +	81	1.6	13 +	3 +	16.4
20–30	143	34	46	80	6	27	30	57	23.77	32.17	55.94	4	20 −	20 +	46 −
30–40	185	67	48	115	11	23	36	59	36 + +	26 +	62 +	5.7	12.4 +	19.4	31.8
40–50	156	38	46	84	11	21	27	51	24.3	29.5	53.8 +	7	15.3 +	17.3 +	36.2 +
50–60	65	19	14	33	4	9	19	28	29.2 +	21.5 +	50 +	6	13.8	29.2 +	44
60–70	23	5	3	8	0	4	11	15	21.7	13	34.7	0	17.3	47.8	65.2
70 up	13	2	2	4	0	4	5	9	15.3	15.3	30.6	0	30.6	38.4	69

This list will help in confirming the first list that astigmatism is caused by the external ocular muscles, by the spherical error influencing the axis of the astigmatism.

COMPOUND HYPEROPIC ASTIGMATISM.

	NO.	PER CENT.	TOTAL
Same sphere, corresponding axis	336	90.4	372
Same sphere, different axis	36	9.6	
Different sphere, different axis	18	75.0	24
Different sphere, corresponding axis	6	25.0	

HYPEROPIC ASTIGMATISM.

Hyper. same cylinder. Ast. corresponding axis	203	90.6	244
Hyper. same cylinder. Ast. different axis	21	9.4	
Hyper. different cylinder. Ast. different axis	38	39.2	97
Hyper. different cylinder. Ast. corresponding axis	59	60.8	
Total carried forward			737

	No.	Per Cent.	Total
Compound Myopic Astigmatism.			
Total brought forward			737
Same sphere, corresponding axis	70	73	96
Same sphere, different axis	26	27	
Different sphere, different axis	76	60.8	125
Different sphere, corresponding axis	49	39.2	
Myopic Astigmatism.			
Same cylinder, corresponding axis	73	92.4	79
Same cylinder, different axis	6	7.6	
Different cylinder, different axis	14	20.2	69
Different cylinder, same axis	55	79.8	
Mixed Astigmatism.			
Converted to same + sphere, corresponding axis	31	77.5	40
Converted to same + sphere, different axis	9	22.5	
Converted to same − sphere, corresponding axis	21	65.9	32
Converted to same − sphere, different axis	11	34.1	
Converted to + and − spheres, corresponding axis	7	46.7	15
Converted to + and − spheres, different axis	8	53.3	
Sphere not same, corresponding axis	13	46.5	28
Sphere not same, different axis	15	53.5	
			1221

DISCUSSION.

DR. J. H. CLAIBORNE, New York City: I am acquainted with Dr. Payne's work on this subject in the past. His paper shows a great deal of thought, deliberation, and patience. I should like to refer in particular to the subject of causation in myopia. Some years ago I read a paper before the eye section of the A. M. A. at New Orleans on the management of myopia, and made a plea for the complete and perfect correction of that refractive error, at least in youth. My paper was based on the views Förster, of Germany, expressed more than twenty-five years ago, in which he combated the idea that myopia is caused by straining the accommodation, and contended that it is caused by the lateral pressure of the extrinsic muscles of the eyeball. Upon this theory, which I consider indeed more than a theory, truly a fact, I have based my treatment of myopia since that time. Therefore, when Dr. Payne speaks of hypermetropia being the cause of myopia I do not agree with him. Some years ago I pointed out that the axis of the convex and the concave cylinders was restricted to certain areas of the circle, as a rule, and I divided the circle into arcs and pointed out the axes of preference of the cylinders within those arcs. I showed that the realm of hyperopic

astigmatism was restricted to an arc of 90 degrees lying between 45 and 135 degrees, and that the axes of selection within this arc clustered around the axis of 90 degrees; that the arc of myopic astigmatism was a small arc of 30 degrees lying between the axes 165 and 15, along the horizontal meridian, and I pointed out that there was a right and left arc of neutrality between 45 and 15 on one side of the circle and 135 and 165 on the other, and that each of the neutral arcs was invaded from time to time by the convex and concave cylinders. I drew three conclusions from the study of the subject which it seemed to me could be defined as laws, as follows: 1. The angles formed by the axis of astigmatism are measured in arcs of a circle. 2. The law of multiples governs the magnitude of these angles and arcs. 3. The arc of 15 degrees is the common factor of the arc and the angles vary in multiples of this number.*

The subject is pregnant with importance to eye surgeons, since I believe the practice of all of us consists of 75 per cent. of refractive errors. While I do not agree with Gould that refractive errors are the cause of diseases of the eye or diseases of the general system, I am convinced that they exaggerate the symptoms in both cases, and I am particular always to correct the refraction after the subsidence of inflammatory conditions of the eye. I am convinced there is no such thing as chance or haphazard in any of the changes that take place in nature, and I feel certain that the same thing obtains in the occurrence of the astigmatic axis. Change is the law of the forces and facts of nature and implies motion. The law of multiples, as we know, governs the forces of gravity, of momentum, chemical change, etc., in all of which change occurs. It is, reasonable, therefore, that the law of multiples should govern the position of the axis of astigmatism, since astigmatism is a change from the normal spheric eye.

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CONSERVATIVE TREATMENT OF ASTHENOPIA IN CHILDREN.

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During a five years' study of refractive conditions among our school-children, the need of conservative treatment seemed to me most important. Unfortunately, it is a subject which has been somewhat neglected and considered decidedly uninteresting. Both public and private school pupils were treated. A more or less successful attempt was made to keep in touch with these pupils by yearly examinations, watching constantly for any new complications. Office methods were copied in the hospital clinic as closely as conditions allowed. Considerable extra time and pains were taken to give hospital patients as good advice and treatment as possible. Palliative treatment cannot be carried out quickly; it is not possible to dispose of all cases in one or two visits. It requires, first of all, a careful assembling of data in order that an exact diagnosis can be made. To understand the complaint of the child it is quite essential to consult with the parent or the school nurse. Only in this way can we decide, in doubtful cases, whether glasses are necessary or whether some other better alternative treatment will bring the desired result. The children were divided into three groups:

Group 1.—Pupils, usually brought by the school nurse, who had high refractive errors. These children need glasses immediately prescribed in order to preserve vision from getting any worse; also to prevent strabismus. Only by this means can they progress steadily with their education.

Group 2.—Scholars who had a small, refractive error (less than one-half diopter), defective vision of one eye, who sometimes complain of indefinite symptoms. About 75 per cent. of these scholars require glasses. A majority should receive a combined treatment to relieve the eyes and other obvious defects, as adenoids, diseased tonsils, decayed teeth, etc. About 20 per cent. of this group never received glasses.

Group 3.—Children brought to the clinic or office by the parent for an eye examination, not because their vision was found defective, but on account of some slight eye symptoms which suggested eye-strain. They may or may not have been notified that the patient had failed to pass the school test. Very often the pupil comes because the parent has observed that headaches start after studying.

Conservative or palliative treatment can be properly undertaken only when careful tests show the following conditions:

A. A very small refractive error, carefully determined under a mydriatic. A comparative test was always made without drops. In my office two independent estimations of the refraction were made, but in the clinic the retinoscopy was generally not attempted without drops, as it takes too long, and our inexperienced house-officers have rarely had an opportunity to try this procedure.

B. The original test of vision, let us say, disclosed a decrease of one eye only. It was reduced to 20/40, at least, in order to have attracted the teacher's attention. The school tests usually were found correct. My own tests tended toward a slightly higher average, because they were more carefully done under a little better conditions.

C. Examinations by the medical or throat consultants at the hospital showed other physical ailments, such as malnutrition, indigestion with constipation, bad teeth, diseased tonsils, and adenoids. Concerning these complications among private cases, it was necessary to get the data from the family physician.

Conservative treatment is justified if we admit that a temporary lowering of vision may accompany the different milder types of chronic conjunctivitis or even hyperemia of conjunctiva. If persistent conjunctivitis is cured with drops, the vision improves and the child may then be able to pass the required visual examination.

This axiom has been one of my guides in the treatment of many children. It is so simple, and at the same time so effective, that often the child requires no further treatment. It seems to me that it should be tried in a great many more cases, but I know that few of my colleagues will agree. The reverse principle has guided most ophthalmologists that, having brought corrected vision to normal with proper glasses, all other ocular complaints would cease. A few of our members will agree that special emphasis should be laid on a principle of conservative treatment, which is directly antagonistic to the views of the majority. It is based on the broad, common-sense idea that glasses for many an average child, of the special group we are considering, should not be the one and only treatment. General practitioners outside of certain larger cities have been guided by this principle. The possibility that these doctors might do harm to the pupils' eyes by ignoring dangerous asthenopic symptoms has been eliminated by the ease with which oculists of nearby smaller towns can be consulted.

Special care was taken to separate the backward scholars in order to give them particular advice. If a child is in its proper grade at school, and complains of indefinite asthenopic symptoms, conservative treatment is indicated. A much more thorough search for the underlying cause of complaint must be made, however, if the child is two or more grades behind the average. It makes no great difference if the scholar is one grade behind the average. A large proportion of pupils in the schools finish their education upon graduating from the grammar school. Several

reasons, aside from physical defects, may explain why they are apparently backward, the most important of which are: A late start, when almost seven years old. A change of residence with a consequent change of school and a readjustment into a different class. Sickness in previous years, but which had left no bad sequelæ to affect the scholar's subsequent progress.

On this account I divided all the children into two groups:

Group A: Pupils in proper grade or one grade behind (84 per cent.).

Group B: Pupils two or more grades behind (16 per cent.).

If a scholar is in Group B, it is the duty of the physician to work with the educators to eliminate as rapidly as possible all physical defects. By so doing a determined effort is made to promote the child to a higher class or at least keep it from falling any further behind. The experience of one of our school nurses is that if a scholar once falls behind, an advancement into a higher class is next to impossible. The best that she expected was to report that the child was holding its own without further complaint, if all medical treatment was carried out faithfully.

Our aim is to improve both the vision and the general ability of the scholar to continue the studies by removing any evident physical defect. Palliative treatment means, of course, that a larger proportion do not receive glasses than in either of the other two groups. This method is used, working on the assumption that so-called eye-strain can sometimes be treated by a combination of general medical treatment and the cure of conjunctival irritation by suitable collyria. A moderate degree of chronic conjunctivitis often proves the most important cause of asthenopia; also a conjunctival irritation, by causing the pupil to think about the eyes, will sometimes disclose a chain of symptoms which have no apparent relation to the eyes. For example, the child complains of the eye aching. This is often described

as a headache. This does not always prove to be a characteristic frontal headache, because small children very often cannot describe accurately this symptom without some further questioning by the doctor. Hemicrania or pain in the eyes seems often the same thing to the patient. To differentiate between the two complaints we must wait until a careful refractive examination has been made before we decide if headache is likely to result from a very small refractive or muscular error. Let us suppose the pupil complains of a frontal headache, with intermittent attacks of pain and tenderness of the eyeballs. This starts two or three hours after the noon meal, at about the time school closes. The headache seems to come after studying. A careful refractive examination is made without coming to any definite conclusion. The diet is investigated, and we discover that constipation and intestinal toxemia have caused the headaches. The medical consultant treats these conditions and the general health of the patient promptly improves. In the meantime soothing collyria having been prescribed, we find often a perceptible improvement of vision. If the first visual test proves the child unable to meet the State requirement, the above treatment will remedy the defect. Mental dulness changes to alertness after freeing the intestines of a number of poisons. At the same time, by relieving conjunctivitis, the child's attention is withdrawn from its eyes, and it proceeds with all studies in a normal manner without further complaint.

If we have found only insignificant physical or ocular defects present, we can now consider a very interesting mental attitude of some scholars toward apparent asthenopia. We realize how easily suggestion will effect a cure of the childish mind. A resignation to the inevitable will also stop all complaints. This is shown very well by observations made by one of the teachers at a large boarding-school for girls in this State. A certain number of the pupils

complained of headache and other asthenopic symptoms and were sent me for examination. For some of the girls glasses were prescribed, and they started again with their studies. It was a rule that only the senior class could study in their rooms, an arrangement much envied by their younger schoolmates. The lower classes all gathered together to study, under the supervision of a teacher, in a large hall. The girls claimed the illumination of this hall was insufficient and that it was a frequent cause of the asthenopia. Most of the younger scholars, for whom glasses had been prescribed, soon became resigned to working under the above conditions and ceased fretting. Also it was noticed that they very often discarded their glasses and made no further complaints. We must acknowledge that any child is liable to act in just this manner. Unless strict supervision by their elders is observed, the child may take off its glasses, especially in cases where hyperopic errors have caused the headache. The vision without glasses being very good, although possibly not perfect, and the headache stopped, the scholar will often experiment to see if it cannot get along without them. The oculist is deluded by thinking that his patient is carrying out his instructions. The ability of the pupil to reëstablish its equilibrium, so to speak, has been ignored. In these cases glasses should not have been prescribed except as a last resort in case all so-called temporary asthenopia could not be relieved by using collyria, or, more often, some general medical treatment.

The need of giving a convex glass to a child to correct temporary accommodation weakness seldom arises. A series of experiments made upon both youths and adults convalescing from pneumonia and typhoid—these diseases chosen on account of the great drain on the patient's vitality—showed a wonderfully quick recovery of the accommodation. Tested one or two days after the crisis of the fever had been passed, the accommodation was generally

reduced to one-half of normal. This reduction was in direct ratio to the patient's youth. Within two weeks the accommodation was again normal. Debility following such serious diseases, however, makes the child complain of eye-strain even from very small refractive errors. The need of giving temporary relief with glasses should not be overlooked. This method of treatment is, of course, universally employed. What is overlooked, however, is a decided tendency of a child to put aside the glasses as soon as symptoms cease, or, anyway, as quickly as experiment shows that no symptoms recur if the glasses are discarded during study hours. As mentioned before, this childish act is inevitable and certainly very often it does no harm. During convalescence from some of the children's diseases, especially measles, the skilful prescribing of suitable collyria will suffice. At such times the child's complaints are apt to be more specific, and we must not fail to give every help to tide the scholar over a difficult period. The length of time that help with glasses or other treatment is needed varies in direct proportion to the child's recuperative power. A neurotic temperament is a complication which prolongs the treatment.

A few scholars are brought by the nurse or mother on account of apparent poor vision of one or both eyes. The failure of the pupil to pass the school tests may be due to shyness or nervousness or to improper illumination of the test-letters. Sometimes my tests did not confirm the previous ones. With such scholars it is often extremely important for the parent to report the actions of the child at home. Attention should be directed to any of the following characteristics: (*a*) Lack of concentration and good application; (*b*) missing entire words or mispronouncing words when reading aloud; (*c*) a tendency to hold the book too close—simulating myopia; (*d*) bad sitting posture; (*e*) eyes or lids inflamed; (*f*) too rapid appearance of fatigue, causing listlessness or inattention.

A temporary decrease of accommodation may follow very vigorous exercises, like football or rowing. Experiments to demonstrate such a condition have been recently begun, and in the future I shall hope to have more information to report.

A small decrease of vision always accompanies the lessened accommodation. This condition is presumably responsible for a good deal of the lack of concentration and inability to do good work during study hours at home in preparing lessons. Such an artificially produced accommodation fatigue of a child may be compared to the easily demonstrated tiring of adult eyes after a busy day. These tests are especially hard to perform, because the child soon lacks interest and then does not make the maximum effort required. The best method seems to be to take a very small number of children and train them sufficiently to observe slight changes of visual acuity. If a series of tests made in the evening show a decrease, control examinations the following morning are necessary. My own observations have proved that my accuracy of observation was influenced often by mental conditions. If the mind is dulled by loss of sleep, nervous fatigue, etc., the test becomes proportionately more difficult. Brain fatigue more often causes a decrease of accommodation than physical exercise.

DISCUSSION.

DR. S. M. PAYNE, New York City: A child with hypermetropia of two or three diopters may refuse glasses for distance. These children always hold the book too close. They do it to magnify the letters. If the doctor will put on convex glasses, the nearer the strength of lens comes to the total amount of the hypermetropia, the further the child will move the book away. The doctor will then see children of eight to ten years of age holding the book off at fifteen inches or more. Correct the full amount if necessary, or give the best glass that increases the distance from the eyes, instead of giving washes, unless they have a blepharitis or something of that sort severe enough to call for treatment.

HEREDITARY CONGENITAL PTOSIS: WITH REPORT OF 64 CASES CONFORMING TO THE MENDELIAN RULE OF DOMINANCE.*

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Near the summit of the Great Smokies, in western North Carolina and eastern Tennessee, on the head waters of Laurel and Nolichucky rivers, have lived for more than a century the strong, sturdy, and virile descendants of one Martin Maney, an emigrant from Dublin, Ireland, a veteran of the Revolutionary War, and one of the first pioneers to cross the Blue Ridge and brave the dangers of mountain wilderness and the Indians of those times—strong and sturdy, as evidenced by their ability to cope with the vicissitudes incident to pioneer life, and prolific, as shown by their large families—18 children in one, 10 to 14 in four, and 28 resulting from the union of one man (No. 17†) to three wives —15 to the first, 2 to the second, and 11 to the third.

Until recently these simple, poor, yet honest mountain people, isolated from railroads and thoroughfares, have lived in this sparsely settled country, where the high altitude, pure water, abundant sunshine, and the many other natural resources are conducive to happiness, longevity, and prolificity. Being content with their natural environment and with their innate love for the mountains, they have not cared to migrate into more populous centers, and on account of their segregation it has been possible to con-

* Candidate's thesis for membership accepted by the Committee on Theses.

† Only 18 of the 28 offspring appear on the family tree because of lack of data concerning remainder.

struct quite an accurate family tree, and to study the condition of hereditary congenital ptosis in many of the 64 cases recorded.

CASE 1.—W. H. (No. 26), aged fifty-one years, farmer, in good health, has double ptosis, but no other evidence of physical or mental degeneration. Forehead greatly wrinkled, especially on right side. Eyebrows standing high. Width of nose, as between nosepiece of eye-glasses, 6 mm.; distance between inner canthi, 29½ mm.; distance between bridge of nose and inner canthus, 11¾ mm.; width of palpebral fissure, 24 mm.; diameter of cornea, 7½ mm.; distance from cornea to inner canthus, 8 mm.; pupillary distance, 55 mm. Upper lid shows no wrinkles, and covers the upper part of the pupil, necessitating constant action of the occipitofrontalis, and tilting of the head backward, in order to fix objects above the horizon. Patient able to superduct eyes, but this to only a limited degree. No other extraocular muscle impairment or imbalance; no pupillary reflex disturbance nor impairment of the ciliary muscles save that due to presbyopia. When thumbs were pressed firmly against the eyebrows, so as to prevent the associated action of the occipitofrontalis, patient was unable to raise the lids, showing total impairment of each levator. Refraction: V.R.E.=0.8, with sph.+0.50⊂cyl.+0.75 axis 15°=1; L.E.=0.8, with cyl.+0.75 axis 165°=1. Presbyopia, 1.50 D.

Patient states that he has never had any trouble with eyes except that due to the ptosis, which is borne out by the fact that he still uses an old cap and ball rifle for squirrel hunting. The patient's son (No. 68), grandson (No. 124), mother (No. 11), grandmother (No. 3), and great-grandmother (No. 1) were similarly affected.

CASE 2.—G. H. (No. 68), aged twenty-five years, son of W. H., resembles in general stature his father, and has about the same amount of ptosis, but is the more able to correct the deformity by the associated action of the occipitofrontalis. Position of head slightly back. Patient able to superduct eyes, but this to only a limited degree. No other extraocular muscle impairment or imbalance; no pupillary reflex disturbance nor impairment of the ciliary muscles.

112 58 113

26

68 124

Deep furrows in forehead, due to muscular attempt to raise lids. Width of nose opposite inner canthi, 11 mm.; distance between inner canthi, 35½ mm.; diameter of cornea, 7½ mm.; pupillary distance, 61 mm. Can lift upper lids slightly above pupil, but, as in case of the father, has no lifting power of upper lids when the action of the occipitofrontalis is checked by pressure over the eyebrows. Can turn eyes upward when lids are lifted, but this action is slightly impaired. Vision each eye, 1. No marks of degeneration.

CASE 3.—C. H. (No. 124), son of G. H., four years old, slightly undeveloped. Has marked ptosis. Upper lids hang below upper margin of pupil, necessitating extreme backward position of head in attempting to fix objects in horizontal field. Small wrinkles in forehead, indicating auxiliary action of the occipitofrontalis. No other extraocular muscle impairment or imbalance; no pupillary reflex disturbance nor impairment of the ciliary muscles. On lifting eyelids with fingers, patient is unable to lift eyes above the horizontal meridian, indicating impairment of the superior recti, but since his father, aunt, and grandfather possess this power of superduction, it is probable that this function will be acquired in later years. Base of nose very broad, measuring 20 mm., compared to 6 mm. of the grandfather and 11 mm. of the father. Distance between inner canthus and side of the nose, 9 mm.; width of palpebral fissure, 20 mm.; height of palpebral fissure, 6 mm.; pupillary distance, 52 mm. Unable to determine vision, but no evidence of defect.

CASE 4.—Mrs. McK. (No. 70), aged twenty-three years, daughter of W. H. (No. 26), has the least amount of ptosis observed in any of the genealogy. Has very narrow nose, like her father; eyebrows highly raised; the skin over the forehead corrugated on account of the contraction of the occipitofrontalis in an effort to assist the levator. Ptosis more marked in the left. On exerting pressure over the supraorbital region, preventing action of the frontal portion of the occipitofrontalis, patient still able to slightly raise the upper lid, showing that the action of the levator in this case is only partially paralyzed. The lacus lacrymalis is very broad vertically, extending downward, inward, and

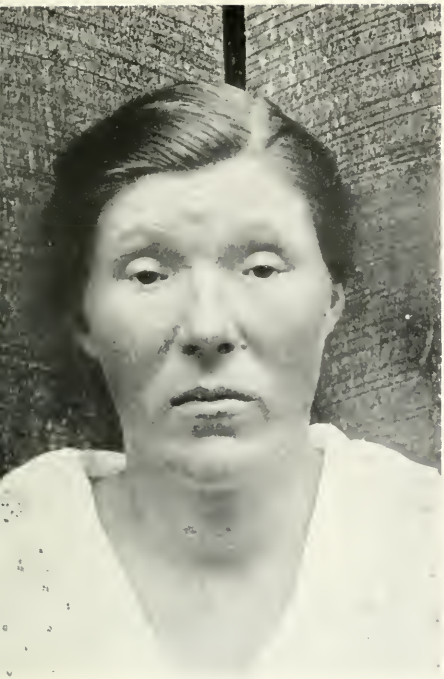
forward. Distance from inner canthus to the side of nose, 8½ mm.; distance between inner canthi, 36 mm.; width of palpebral fissure, 23 mm.; vertical width of palpebral fissure, 6½ mm.; pupils, in moderate light, 3½ mm.; crest width of nose, 14 mm.; pupillary distance, 64 mm. On looking straight forward, upper lid covers upper part of pupil. On attempting further upward motion the occipitofrontalis is called more and more into action. Vision above 20 degrees impossible without bending head backward.

CASE 5.—Male infant (No. 81), six weeks old, in splendid health, has very marked ptosis. There is no evidence of action of the occipitofrontalis as an auxiliary aid to the levator, and, as the levator has no action, the lids scarcely separate, there being perhaps about 1 mm. opening. Palpebral fissure very narrow to the extent that blepharophimosis exists. Lifting upper lids causes immediate photophobia, but reveals eyes normal in appearance. Unable to test extrinsic muscles, but no evidence of their impairment.

CASE 6.—(No. 111.) Girl, aged fifteen years, in good health, moderate amount of ptosis; total impairment of function of levators. Has congenital contraction of the subcutaneous tissues over the malar, or attached to same, drawing the lids at the outer canthus, especially the lower, far away from the eye. This contraction of the lower lid stretches the lower canaliculus, misplacing the lower punctum 7 mm. from the inner canthus, and gives a general appearance to the eyes of convergent strabismus, but this is only apparent, as there is no muscular imbalance. There is a chronic granular condition of the lids, due to their exposure, and the cornea in the lower outer quadrant, for the same reason, is roughened, rendering the vision 0.5 in the right eye, that in the left being normal.

The six cases above reported represent the fourth, fifth, and sixth generations. Cases Nos. 81 and 124, infants in the sixth generation, have seemingly an equal if not a greater amount of ptosis than had case No. 26 or case No. 58 in the fourth generation, or of any shown in any of the photographs, which would indicate that the degree of ptosis remains constant, and is at least not becoming diluted.

SUMMARY.—(See Chart No. 1.) The cases of hereditary



43

111

70

46 88 87

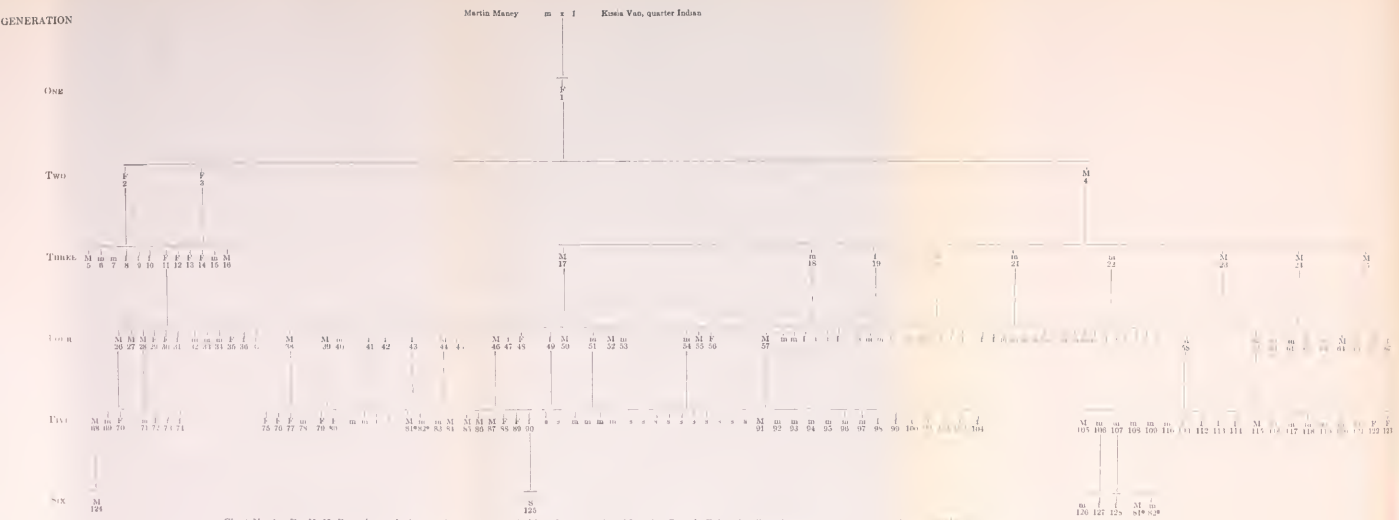
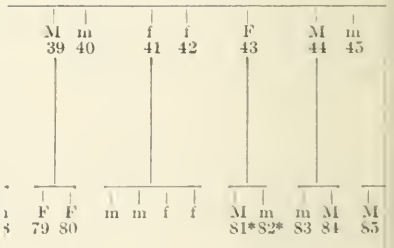


Chart No. 1.—Dr. H. H. Briggs' genealogic tree of cases of congenital hereditary ptosis. M, male affected; F, female affected; S, sex unknown, affected; m, normal male; f, normal female; s, sex unknown, normal

*Offspring M 81 and m 82 resulted from union of second cousins, m 109 and F 43.



H. H. Briggs' genealogic tree of cases

ptosis herein recorded are members of a genealogy extending through six generations over a period of one and one-quarter centuries. Inheritance in every case is direct, no generation in the lineage of the affected being skipped except in the one instance, Case No. 125, in the sixth generation, whose mother, No. 90, a member of the genealogy, is said to be normal, and to have married one who was also normal.*

There was only one case of intermarriage,† and that between affected female No. 43, in generation four, to a normal male, second cousin, No. 109, in generation five. Of the two male offspring, one was affected and the other normal.

Twenty-three families are represented, in 17 of which the father, and in 6 the mother, transmitted the malformation. In no case did affected parents fail to transmit ptosis to one or more of their offspring.

Generation	Number Members			Affected			Normal			Affected Parents		
	Male	Female	Total	Male	Female	Total	Male	Female	Total	Male	Female	Total
I	..	1	1	..	1	1	..	..	..	..	1	1
II	1	2	3	1	2	3	..	..	..	1	2	3
III	13	8	21	6	4	10	7	4	11	5	1	6
IV	27	15	42	16	7	23	11	8	19	9	1	10
V	31	25	56	9	16	25	22	9	31	2	1	3
VI	4[1]	2	7	2[1]	..	3	2	2	4	..	..	..
Total	76[1]	53	130	34[1]	30	65	42	23	65	17	6	23
Enumerated twice	2	..	2	1	..	1	1	..	1	..	..	..
	74	..	128	33	..	64	41	..	64	..	..	..

[1] One sex unknown.

Of the 128 members of the genealogy composing the 23 affected families, 64 were affected with ptosis and 64 were

* Information gained from relatives.

† Mountain people seldom intermarry.

normal; 74 were males, 53 females, and one of unknown sex. An equal number (64 of each) were affected and normal. Of the affected, 33 were males, 30 were females, and 1 of unknown sex, while of the normal, 41 were males and 23 females.

Of the twenty* affected parents, 16 were males and 4 females. Of the 121 offspring (71 males and 50 females), 61 were affected and 60 normal. The 16 fathers had 100 children (62 males and 38 females), to 47 of whom they transmitted ptosis, there being 53 normal. The four mothers bore 21 children (9 males and 12 females), of whom 14 had ptosis and 7 were normal. Of the offspring, 61 had ptosis and 60 were normal, the fathers' share was 47 ptosis to 53 normal, while the mothers' was 14 ptosis to 7 normal. Of the 71 male offspring, 62 were from affected fathers and 9 from affected mothers, while of the 50 female offspring 38 were from affected fathers and 12 from affected mothers.

	PARENTS	MALE	FEMALE	PTOSIS	NORMAL	TOTAL
Fathers	16	62	38	47	53	100
Mothers	4	9	12	14	7	21
	20	71	50	61	60	121

Literature.—Ptosis unaccompanied by motility defects of the eye is seldom seen. Epicanthus and impairment of function of the superior rectus most frequently exist, yet paralysis of each and all of the extrinsic muscles has been noted. In no case found in the literature has paralysis of the intrinsic muscles of the eye been a complication of ptosis, except in complete ophthalmoplegia. Disturbance of the pupil and accommodation are, therefore, practically never seen associated with ptosis.

* Leaving off the progenitor, father of No. 1, parents who intermarried, and No. 90 (mother of 8), from total number of parents.

Hirschberg[17] found a case of ptosis accompanying total ophthalmoplegia in a child whose mother and grandmother were similarly affected. Schiler[35] reports father and two sons having ptosis with paralysis of all the outer eye muscles. Horner[19] reports inherited ptosis through three generations of one family, and in nine brothers of another family, associated with impairment of the superior rectus. Rampoldi[33] reports ptosis in father, son, and daughter, with immobility of the extrinsic muscles and slight astigmatism. Daguillon[9] reports congenital hereditary ptosis with divergent strabismus, congenital hereditary myopia, and amblyopia. Lawford[25] reports ptosis in father and three sons among seven children, immobility of the eyes in the vertical meridian, with only slight amount of mobility to the side. Horner[19] reports cases of ptosis in several generations of the same family. Heuck[16] reports ptosis in mother, two sons, and one daughter, in which the eyes were directed downward and easily converged. There was complete loss of function in the superior and inferior recti, while the other extrinsic muscles functionated. There was also deficient vision. Hirschberg[17] reports ptosis in a mother, daughter, grandson, and great-grandson, with impairment of superduction, abduction, and adduction. In the grandson, epicanthus, superduction impossible, and divergence alternating with convulsive convergence. Vignes[43] gives cases of ptosis and epicanthus in grandfather, five sons, and one daughter of 11 children. He also reports ptosis in a father, two sons, and one daughter of 5 children. Vossius[44] gives history of two brothers with impossible movement of the eyes in the vertical meridian and dimness of vision. Ginestous[12] reports child of twenty-five months showing incomplete ptosis, epicanthus, and paralysis of the superior rectus. Ahlström[1] reports case of ptosis and ophthalmoplegia externa in a fifteen-year-old boy, and history of several relatives similarly affected. Guende[15] reports three brothers in

family of nine children with ptosis complicating ophthalmoplegia externa. Dujardin[11] reports maternal grandfather, mother, and four daughters having ptosis with immobility of the eyes upward and downward and limitation of lateral movements. Schiler[35] reports ptosis in grandfather, father and son, with immobility of the outer muscles, high hypermetropia, and a dot-like darkening of the lens. Gourfein[14] reports ptosis in grandfather, father, and four sons, the female members of the second and third generations remaining healthy. Movements of the eyes always accompanied by rotatory nystagmus. They also showed amblyopia, changes in the optic nerve and the retina, flattening in the region of the eyebrow. Ayres[4] reports grandfather and uncle with immobility of all eye muscles. Paul Bloch[7] reports two brothers having paralysis of the abducens and ptosis.

Steinheim[39] reports ptosis and epicanthus in five generations (see Chart No. 2). The great grandfather had ptosis. Of his five children, two males and one female were affected. One male and one female married and moved to America, no more being known of their descendants. The other male married, had two daughters, one normal and one affected; the latter becoming the mother of five children, of whom two males and one female were affected. Of the girl's children two were normal and one affected. Of the two males, one became the father of two normal and two abnormal children, while the other was father of three abnormal and three normal offspring.

Hüttemann,[21] in 1911, reported 11 cases of congenital hereditary ptosis and epicanthus in three generations of the same family (see Chart No. 3). In all these there was a peculiar lid movement associated with lateral movement of the eyes. In a glance to left the right eye, and on looking to the right the left eye, was almost closed.

Dujardin[11] reports five cases of congenital ptosis in grand-

father, mother, and three daughters, the fourth daughter being normal. One of the affected daughters had partial ophthalmoplegia (see Chart No. 4).

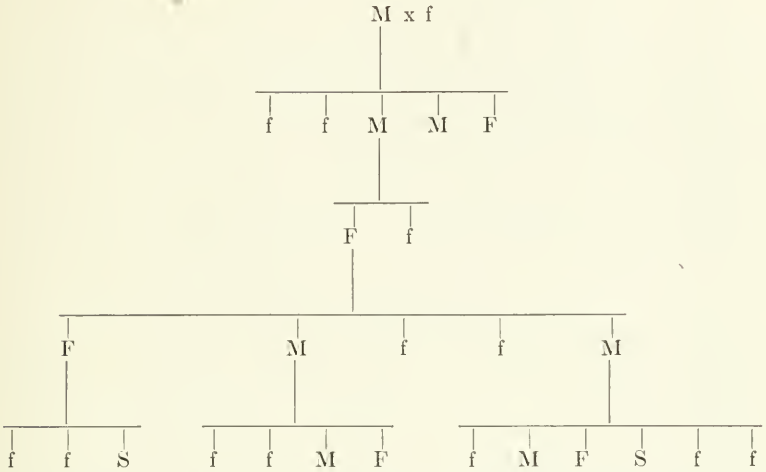


Chart No. 2.—Family tree of Steinheim's thirteen cases of hereditary ptosis and epicanthus. M, affected male; F, affected female; f, normal female; S, abnormal, sex unknown.

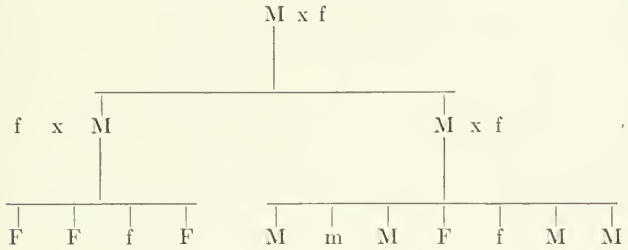


Chart No. 3.—Family tree of Hüttemann's eleven cases of hereditary congenital ptosis and epicanthus. M, affected male; F, affected female; m, normal male; f, normal female.

H. P. Stuckey[40] reports father, son, and three grandsons living in northern Georgia having ptosis, one daughter being normal. These are Nos. 25, 64, 65, 66, and 67 of the genealogy of the author's cases. He considered it a probable

example of Mendelian recessive. The inheritance is, however, dominant in character, as is amply illustrated in the extensive family tree.

F. R. Spencer[37] reports a case in a man of twenty-seven who gives history of ptosis since fifteen or sixteen years of age. He thinks it has gradually increased since that time, but has been much worse for the past three years. His mother, maternal grandmother, two maternal aunts, and one brother were similarly affected. His younger brother's lids began to droop at the age of twenty-two years. His mother had great difficulty in raising her lids. His abduction, adduction, superduction, and subduction were very

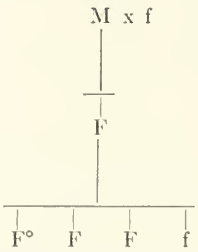


Chart No. 4.—Family tree of Dujardin's five cases of congenital ptosis, with partial ophthalmoplegia in F°. M, affected male; F, affected female; f, normal female.

limited, and he was unable to raise either eyelid without the assistance of the frontalis muscle.

Pathology.—Operations, autopsies, and microscopic examinations have revealed the following conditions as causes of ptosis:

(*a*) Defective development of levator and other muscles of the eye. Heuck[16] found a partly developed levator measuring only 2 mm. in breadth. Bach,[5] in a case of bilateral congenital ptosis and limitation of the eye movements upward, found defective development of levator and moderate atrophy of superior rectus; the nuclear region of the oculomotor was normal.

(*b*) Adhesion of Muscles.—Albers and Wrisburg[2] found in a case of ptosis adhesion of the rectus superior with the levator; the external rectus was adherent to the inferior rectus, and the internal rectus to the superior oblique.

(*c*) Abnormal insertion of muscles was found by Rossi,[34] Heuck,[16] Dieffenbach,[10] Pflüger.[32] In some of the cases the superior rectus was found inserted back of the equator.

(*d*) Connective-tissue bands instead of muscles were found by Ahlström[1] in cases of congenital ptosis. When he laid bare the tarsal border in the left eye, no trace of the levator tendon was seen, and in the right eye only a few scattered tendon fibers were found.

(*e*) Absence of Muscle.—Lawford,[25] in a case of ptosis with divergence, found the rectus internus absent. Ahlström[1] found no trace of the levator, and Heuck[16] reports finding the same condition. Horles[18] reports a case of ptosis in which both obliques were lacking, the recti being normal. Seiler[36] found a case of ptosis in which the inferior oblique and superior rectus of the right eye and the inferior oblique of the left eye were lacking. In another case the superior and inferior obliques of the right and the superior and inferior obliques and superior rectus of left were not found.

Steinheim,[38] in a case of congenital ptosis with defective motion of the eye, failed to find the superior rectus.

Etiology.—The etiology of ptosis leads back to the question of the cause of variation, and has been defined as the event which brings about the addition or omission of a factor. It is due either to absence of the factor for the normal development of the levator, or to the presence of an inhibitor to normal development.

Intermarriage, certainly in the last six generations, has not been a factor in the heredity of ptosis in this genealogy. Of the 128 offspring whose histories have been studied, there has been but one intermarriage, that between the affected female (No. 43) in the fourth generation and her second

cousin, normal (No. 109) in the fifth generation. From this union resulted one normal male, three years old, and one male six weeks old.

There is one other instance of intermarriage between female (No. 36) and son of No. 21, who was grandson of No. 4, abnormal, two generations removed, and consequently not included in the numbered offspring. This union resulted in all normal.

It is very difficult to determine the exact anatomic cause of the anomaly. Because of their anatomic situation, it is impossible to test the muscles of the eye electrically. Koster[23] reasoned that, if the enlargement of the palpebral fissure is considerable, the levator muscle of the lid exists but is paralyzed or atrophied. Since the tarsal muscle of Müller is inserted in the lower part of the tendon of the levator, if the latter is lacking, the tarsal muscle has no point of insertion and cannot raise the lid under the influence of cocain. Cocain, therefore, reveals the absence or presence of the levator. The use of this method in the author's cases examined, resulted, so far as revealing a cause, negatively.

The author thus far has not had an opportunity of operating on any case of ptosis in this genealogy, nor to note the exact pathologic cause of ptosis at necropsy. He hopes, however, to operate on case No. 111, and to soon make a supplemental report of the findings.

Diagnosis.—Ptosis is either congenital or acquired, and these again may be subdivided according to the causes which produce it. It may also be unilateral or bilateral, although some hereditary eye diseases, such as certain forms of cataract (Nettleship[31]) which occur in middle life, are not congenital. All cases of hereditary ptosis are congenital. In other words, hereditary ptosis is prenatal, manifesting itself at birth and continuously. In hereditary congenital ptosis, which is usually bilateral, there may be absence or deficiency of the levator often associated with absence or deficiency of

one or more of the extrinsic muscles of the eye, most frequently the superior rectus, and most frequently of all it is associated with congenital epicanthus. There may be absence or deficiency of that portion of the third nerve supplying the levator. It is sometimes associated with sympathetic movements of the lower jaw, increasing in degree when the eye is abducted, and disappearing when it is adducted, the lid in these cases constantly retracting involuntarily when the jaw is moved to the opposite side of the ptosis (Bradburn[8]).

On the contrary, in an acquired ptosis there is usually to be found affection of the lids, such as hypertrophy, edema, new-growth, blepharochalasis, and ptosis adiposa. It may result from trauma to the levator, or atrophy, as occurs in women of middle life, or as an early symptom of chronic progressive ophthalmoplegia and myasthenia gravis. It may result from a central or cerebral degeneration. When due to a cortical lesion, it would probably be unilateral, and the only eye lesion, the other branches of the oculomotor nerve being unaffected. A nuclear lesion of the pons might produce such lesion (Bradburn[8]). Ptosis, as a symptom, may result from constitutional diseases, such as myasthenia gravis, locomotor ataxia, toxic processes, chronic progressive ophthalmoplegia, hysteria. Acquired ptosis may also occur from nuclear, fascicular, or basal lesions, from sphenoidal abscess and orbital lesions, and from affections of the sympathetic nerve.

The author's cases of ptosis are unique in that in no case is the ptosis complicated by any other motility defect except in the infant (No. 124), whose superior rectus did not functionate, and this probably not because of any defect in the muscle itself or impairment of its nerve supply *per se*, but on account of the drooping of the lid the superior rectus had never been called upon to turn the eye upward. When the child was asked to look at an object held above his horizon,

he invariably tilted the head backward until the visual plane met the object, and this he did even when the lids were lifted by the observer. Observation of the eyes during sleep or under general anesthesia might have determined whether or not superduction was possible, but such opportunity was not offered.

In case No. 59 it was demonstrated that the levator had a certain amount of function, but in no other case examined could function of the levator be demonstrated.

Mendel's Theory.—The Mendelian phenomena are well illustrated in peas, mice, rabbits, poultry, and snails. Certain defects: hornless cattle—polled angus, earless sheep of China, tailless cats of Japan, short-tailed dogs and pigs are also examples of Mendelian phenomena. That we do not find more examples of Mendelian inheritance in man is due to the fact that the inbreeding necessary to bring out Mendelian segregation is not sufficiently close in man.

The eye and its appendages are subject to more hereditary diseases and malformations than is any other organ, due partly to the great number of histologic and anatomic structures concerned in its makeup, and partly to its many complicated and coördinated physiologic functions, which are so easily disturbed. Of ophthalmic diseases most frequently transmitted may be mentioned: ptosis, epicanthus (Manz[27]), distichiasis, nodular and reticular opacities, corneal staphyloma, aniridia, coloboma of the iris, corectopia, cataract (Nettleship[31]), ectopia lentis, glaucoma, retinitis pigmentosa (Leber[26]), and other retinal degeneration, night-blindness, nystagmus, color-blindness, albinism, motility defects, and errors of refraction. Of these, those showing the most marked evidence of transmission are cataract, night-blindness, color-blindness, and motility defects, including ptosis. Most of the transmitted defects of the eye follow Mendelian rules, and, with the exception of those which are sex-linked,

as color-blindness and stationary night-blindness, are *dominant* to the normal.

In order to discuss more intelligently the inheritance in the cases of ptosis herein reported, let us recall briefly the salient points of the Mendelian theory.

Gregor Mendel,[28] in 1865, experimenting with the edible pea (pisum sativum), took a pair of characters, tallness and shortness, and crossing the tall variety of six feet with the dwarf of one foot, it was found that the first cross-bred variety, F 1, were all tall. From the fact that the character tallness appeared in all the cross-bred, to the exclusion of the opposite character, dwarfness, Mendel called it a *dominant* character, and dwarfness a *recessive* character. The tall, cross-bred by self-fertilization, bore seeds which produced a mixed generation, F 2, many being tall and some being short, like the tall and short grandparents, respectively, and in the ratio of about 75 per cent. tall and 25 per cent. short. The F 2 plants were again allowed to self-fertilize themselves, and it was found that the dwarfs (recessives) produced dwarfs entirely, and that further propagation of these produced pure recessives. But the tall, F 2, dominants, produced (instead of all dominants as the dwarfs produce recessives) two kinds: (*a*) plants of mixed F 3, consisting of tall and dwarfs in the proportion of three to one respectively, and (*b*) plants which gave tall only, and are those pure to tallness; the ratio of the impure (*a*) plants to the pure (*b*) plants being two to one. The F 2 generation dominants was composed, therefore, of three kinds of plants:

$$3 \begin{cases} \textit{Dominants}, \text{ pure, } 25 \text{ per cent.} \\ \textit{Dominants}, \text{ impure, } 50 \text{ per cent.} \end{cases}$$
$$1 \quad \textit{Recessives}, \text{ pure, } 25 \text{ per cent.}$$

Mendel found similar inheritance as above noted when all the other distinct characters in peas were used, the dominant character being shown in italics as follows: *Tallness*—

shortness. *Flowers along axis* of plant or on top of plant. *Green color of unripe body*—yellow color of pods. *Shape of body inflated*—constricted between seeds. *Red seeds*—gray or brown. *Yellow cotyledons*—green. *Round seeds*—wrinkled.

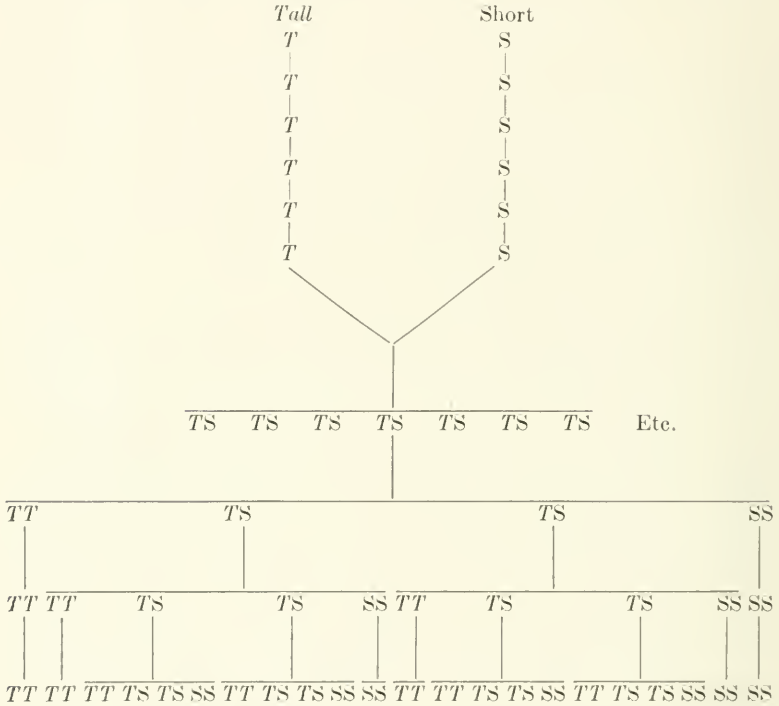


Chart illustrating Mendelian inheritance of tallness (dominant) and shortness (recessive) in the edible pea (pisum sativum). *TT*, Represents pure dominant, SS pure recessives, while the *TS*, *TS*, *TS*, etc., are hybrids. The character tallness is manifest in dominants and hybrids, and is indicated in italics. The character shortness is manifest only in SS shown in Roman type.

Segregation and allelomorphism. Tallness (D) and dwarfness (R) (Bateson[6]) entered in the fertilized ovum in the original cross, but since the next generation showed some tall and others dwarfs, there must have been a separation (segregation) of the two characters in the process of the germ

formation in the fertilized ovum (zygole) of the two characters, the segregated characters being called allelomorphic. This was shown in crossing F 1 with pure dominants and pure recessives. D R multiplied by D D gave all dominants in appearance, although composed of D R and D D plants equal in number on the average. On the other hand, D R multiplied by R R gives an equal number of dominants and recessives; the dominants being D R and the recessives being all pure recessives. This ratio of one to one, which results from D R multiplied by R R, will be referred to later in relation to the inheritance of ptosis.

If the character ptosis is dominant, the normal being recessive, there should be, in the aggregate, approximately the same number of affected and normal offspring. There were found 64 cases of ptosis and 64 normals, which conforms with mathematical precision to the rule of dominance.

In this connection it is interesting to compare the ratio of affected and normal in cases of hereditary cataracts taken arbitrarily from Nettleship's reports, as follows:

RECORDER	AFFECTED	UNAFFECTED
Berry	20	20
Fukala	19	22
Nettleship	29	25+
Zirm	14	15
Nettleship and Ogilvie	17	25
Total	99	107

Dominants.—The following points, quoted from Bateson,[6] differentiate between dominants and recessives. "Dominant characters will, in general, be recognized as such from the fact that they are transmitted through affected persons only. The dominants will, as a rule, have one parent affected with the peculiarity and one parent free from it. It is then to be expected that the children of such dominants, resulting from their marriages with unaffected persons, will show equal numbers of affected and normal. Recessive characters will be recognized by the fact that they may appear in the chil-

dren of parents not exhibiting such characters, and especially among children born of consanguineous marriages. Complete proof of the recessive nature of a characteristic will be obtained only by evidence that all the children of affected parents exhibit the characteristic."

From this we reason that the author's cases conform to the Mendelian law of dominance, because—

(*a*) The transmission is through affected persons only; there being no case where the inheritance was through an unaffected parent, except in case No. 125, a child whose mother is said to be the only normal of seven children. As this information was obtained from relatives, it is entirely possible that a slight degree of ptosis in her case might be considered normal compared to the marked degree which the other six are reported to have.

(*b*) In every case the dominants (ptosis) have one parent affected and the other parent normal. There is only one case of consanguineous marriage among the cases reported; that being between an unaffected male in the fifth generation (No. 109), wedded to an affected female (No. 43) in the fourth generation; the relationship being that of second cousins, and resulting in one affected and one normal offspring, according to the rule of dominance.

(*c*) The ratio of 64 dominants (ptosis) to 64 recessives (normals) conforms to the third qualification of dominants which requires an expectancy of an approximately equal number of normal and affected children descending from dominants (ptosis).

(*d*) That it is not a recessive character is shown by the fact that in no case was an affected child born from normal parents, one or the other parent invariably showing the character, except in the one case, No. 125, and as this information was gained from relatives, there is a possibility of it not being correct.

(*e*) Thus far there has been no opportunity to determine

what would be the result in the offspring where both parents are affected.

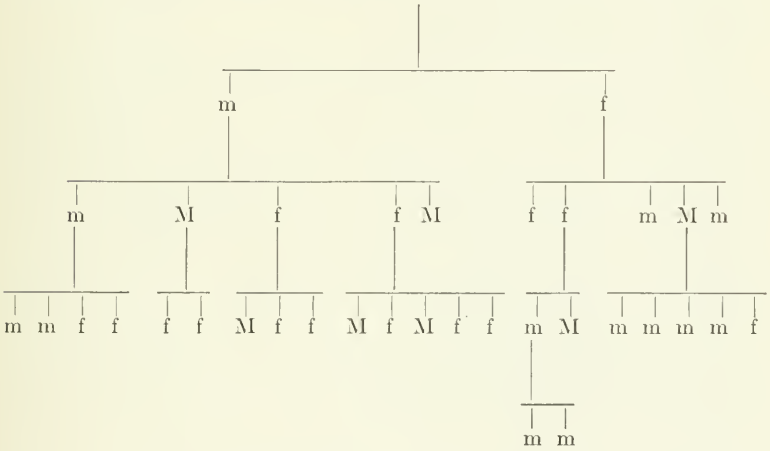
Sex-linkage.—Certain ocular diseases of the dominant type follow a sex-limited descent, notably color-blindness and night-blindness. The distinguishing differences in the sex-limited, from the pure dominant types of inheritance, are that in the former—

(*a*) The males are affected more frequently than the females.

(*b*) It may be transmitted by affected males, but never by the unaffected males.

(*c*) It is transmitted by unaffected females.

(*d*) Apparently normal women, daughters and sisters of affected males, transmit the condition to their sons. The accompanying pedigree illustrates the sex-limited descent of color-blindness in a family found by Dr. W. H. R. Rivers (Bateson[6]) among the Todas, a hill-tribe of southern India.



Family tree of Dr. W. H. R. Rivers' cases of color-blindness among the Todas, a hill-tribe of southern India.

In no case in the genealogy save two was an affected child born to a normal parent. These are case No. 1, the first

member of the genealogy, an affected female resulting from the union of Martin Maney, an Irish descendant, wedded to Kissia Van, a quarter Indian, both of whom had normal eyes. These cases were not examined, as they lived in the latter part of the eighteenth century, but this information comes from so many reliable sources, and especially from the oldest inhabitants in the neighborhood, that its correctness can scarcely be doubted.

Quoting from a letter from Dr. I. L. English, who was reared in that country and has been the only physician practising there: "Martin Maney, who came when a boy from Dublin, Ireland, married an Indian. John Metcalf (No. 24) thinks that Martin Maney was little-eyed, but I learn from other old people that he was not; that when his first child (female No. 1) was born, little-eyed, the friends made alarm about the child's deformity, and its father (Martin Maney) told them to not worry, that most all his people in Ireland were little-eyed." This is the first instance of an affected child born of normal parents.

The second case is that of No. 125, sex unknown, said to be affected, born of normal female (No. 90). This family lives in middle Tennessee, and I was unable to verify the statement. It is quite possible that in a family where five of the six children have ptosis, the sixth child might, by contrast, appear normal, when in reality it might have a moderate degree of ptosis. Granting that the information is correct, it would probably mean an instance of recessiveness such as perhaps happened in the case of F 1, the first member of the genealogy. Except in these two cases the inheritance is purely dominant in character.

INDEX TO GENEALOGY.

m. Martin Maney
f. Kissia Van
1.* Bettie Maney Metcalf
2.* Bettie Metcalf Hensley
3.* Kissia Metcalf Barrett
4.* Hiram Metcalf
5.* Hiram Hensley
6. Joe Hensley
7. William Hensley
8. Rena Hensley
9. Nancy Hensley
10. Lucinda Hensley
11.* Lucinda Barrett Hensley
12.* Lucinda Barrett
13.* Levina Barrett
14.* Jane Barrett
15. Hiram Barrett
16.* Jack Barrett
17.* Absalom Metcalf, second
18. Demps Metcalf
19. Lucinda Metcalf Hensley
20. William Metcalf
21. Henry Metcalf
22. Wesley Metcalf
23.* Enos Metcalf
24.* John Metcalf
25.* Cling Metcalf
26.* Woods Hensley
27.* Jack Barrett
28.* Hiram Barrett
29.* Jane Barrett
30.* Cenia Barrett
31. Rena Barrett
32, 33, 34, 35.*
36. Sue Barrett
37.
38.* Lafayette Metcalf
39.* Fred Metcalf
40. Shown Metcalf
41. Cora Metcalf
42.
43.* Elsie Metcalf
44.* Fletcher Metcalf
45. Kenneth Metcalf
46.* Hiram Metcalf
47. died in infancy
48.* Nancy Jane Metcalf
49. Cornelia Metcalf
50.* Joe Metcalf (unmarried)
51. Gus Metcalf
52.* Tom Metcalf (unmarried)
53. Shubert Metcalf (unmarried)
54. John Franklin Metcalf
55.* died in infancy
56.* Emma Metcalf (dead)
57.* Willard Metcalf
58.* Brownlow Metcalf
59.* Enos Henry Metcalf
60. Sam Metcalf
61. Gus Metcalf
62.* Sophronia Metcalf
63. William Metcalf
64,* 65,* 66,* 67.
68.* Garrett Hensley
69.
70.* Mrs. McKinney
71, 72,* 73, 74, 75,* 76,* 77,* 78,* 79,* 80,* 81,* 82, 83, 84,* 85,* 86,* 87,* 88,* 89,* 90, 91,* 92, 93, 94, 95, 96, 97, 98,* 99, 100, 101, 102.
103. died in infancy
104.
105.*
106. Arthur Metcalf
107. Hubert Metcalf
108.
109. Charlie Metcalf
110.
111.* Kitty Metcalf
112.*
113.*
114.*
115.* John Metcalf
116. Raburn Metcalf
117. Joel Metcalf
118. Roscoe Metcalf
119. Millard Metcalf
120. Oscar Metcalf
121. Thomas Metcalf
122.* Della Metcalf
123.* Sue Emma Metcalf
124.* Clyde Hensley
125.* sex unknown
126, 127, 128.

Numbers marked with a * represent members of genealogy affected with ptosis.

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A CONSIDERATION OF THE CAUSES OF HETEROCHROMIA IRIDIS, WITH SPECIAL REFERENCE TO A PARALYSIS OF THE CERVICAL SYMPATHETIC.*

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That curious condition which consists in a difference in the pigmentation of the two eyes is regarded by the casual observer as a play or caprice of nature. This phenomenon has for centuries been noted, and was called *heteroglaucus* by Aristotle.[1] One who seriously studies the subject is at once impressed with the complexity of the situation and soon learns that nature plays a comparatively small part in its causation. It is, however, only within a comparatively recent time that the pathologic aspect has been considered, and in this discussion I especially wish to draw attention to that part played by the cervical sympathetic.

Many writers have given Hutchinson[2] (1869) credit for first describing a difference in the color of the irides, in which cataract was associated. By chance I discovered that Lawrence[3] (1853) described irregularities in the color of the iris and cited two cases, in one of slowly changing color and another of changed color of the iris with cataract. He also quoted Wilde,[4] who mentions the fact that heredity plays an important part in many of these abnormalities.

Nomenclature.

Various terms have been applied for this inequality in the color of the irides, such as heteroglaucus of the ancients;

* Candidate's thesis for membership accepted by the Committee on Theses.

heterophthalmus (Weill[5]), aniso-iridochromia (Scalinci[6]), and chromoheteropia (Malgat[7]). The last named thinks that the word heterochromia should apply to those cases in which parts of the same iris have different colors. In those cases where a cyclitis accompanies the iris decoloration Butler[8] uses the term "heterochromic cyclitis," but the "chronic cyclitis with decoloration of the iris," as described by Fuchs,[9] undoubtedly gives a more accurate description of the disease, notwithstanding its long title. The commonly accepted and most universally used term, *heterochromia iridis*, exactly expresses and implies the picture from its derivation (ἕτερος, other; χρῶμα, color). Other newly coined words only add disorder to an already chaotic classification of diseases.*

Classification.

Any exact classification of this condition is almost an impossible task, and, mindful of its limitations, I venture to submit the following as a working basis of study:

Primary	Congenital	with or without	Heredity. Sympathetic paralysis. Cyclitis and cataract.
	Acquired	with or without	Sympathetic paralysis. Cyclitis or cataract.
Secondary			Injuries. Siderosis bulbi. Uveal infection—Myopia. Glaucoma.

Strictly speaking, as Sym has pointed out, there could be no such thing as a congenital form of heterochromia iridis, since it is about the twelfth or sixteenth month that the

* Dr. Willis H. Bocock, my friend and former professor of Greek at the University of Georgia, has suggested the word *irideterochromia*. "The *o* (as in iridoplegia) is not used in composition before a vowel; therefore *irid* in the word above. *Hetero* (as in heterochromia) loses the *h* in composition, therefore *irideterochromia*."

While it would be advantageous to have the meaning so expressed in one word, its derivation would not be so obvious as the term heterochromia iridis, unless the reader possessed a working knowledge of Greek.

child's iris ceases to be light and takes on color. Brown pigment has, however, been noted to appear on the iris as early as six months.

I am not in accord with Sym[10] and Scalinci, who maintained that we should reserve the term heterochromia iridis "for the instances in which there has not been any obvious pathological process in the eye, such as glaucoma or iridocyclitis." The term expresses the condition regardless of the cause. I have recently seen a case in which there was a decoloration of the iris in an adult negro who, at first glance, presented evidences of a sympathetic paralysis, namely, a smaller pupil and a narrower palpebral aperture than in the opposite eye. The case proved to be the result of an injury received during childhood, and the presence of a foreign body in the choroid could be demonstrated. The ptosis was traumatic, the myosis was due to posterior adhesions, and the iris decoloration was probably a siderosis bulbi, or caused from a low-grade iridocyclitis. I can see no objection to classifying this as one of heterochromia of the iris of secondary origin, due to an old injury (foreign body with uveitis).

Heredity.

Heredity as a cause for heterochromia iridis has not received the same consideration from modern writers as from those of a generation ago. I am firmly convinced that there are many such congenital cases without sign or development of a pathologic lesion; and one reason why more cases are not so classified is that the observer, indifferent to the causes of heredity, does not stress its influence, and also that too few of our patients, clinical patients especially, know enough of their ancestry.

The history of cases 1 and 2 from the same family (more fully described under the heading of sympathetic paralysis), where the paralysis and lighter iris were all on the same side, in a manner substantiates the fact that heredity does

play an important part. If the paralysis was the cause for the iris decoloration, it would be proper to consider it as the disorder inherited.

Galezowski,[11] Bistis,[12] and Scalinci[6] report cases of sympathetic paralysis where one or more children in a family had iris decolorations which were inherited. But we should bear in mind Priestly Smith's[13] words that "heredity is not in any real sense a cause. It may account for the presence of a disorder in an individual or family, but it tells us nothing as to its origin. In some former generation there must have been a first departure from the normal."

Osborne,[14] as early as 1849, drew attention to the difference in color of the irides of a person (that is, they were irregularly marked) who had 15 brothers and 5 sisters who all possessed the same abnormality which was derived from the mother, whose three sisters and one brother had the same, which were again derived from the mother's side. Gunn[15] mentions similar cases and classifies them as "piebald iris."

It has been stated many times that in congenital heterochromia iridis the color of one eye is inherited from the father, while the color of the other is derived from the mother. In many instances this has been true, but the reverse is as often the case, and even eyes of an entirely different color to either parent have been observed. This contradictory observation, however, would not refute the hereditary theory of transmission, bearing in mind the principles of Mendel's law.

The family reported by Gossage[16] is the only recorded case beyond the second generation that I find mentioned. A detailed description and examination of the eye unfortunately is not given, although the author states that "in this family heterochromia of the iris tended to appear when one eye, always the left, was grayish blue in color, with chestnut-brown patches. Of the offsprings of the affected

members of the family, eight were affected and twenty-two free" (Fig. 1).

From a genetic point of view, Przibram's[17] work on Angora cats is interesting. He found that the union of an asymmetrically colored animal having one blue and one yellow eye, with a symmetrically colored animal having two blue or two yellow eyes, resulted in the production of both asymmetrically (heterochromia iridis) and symmetrically pigmented offsprings. He concluded that asymmetric animals can be traced back to asymmetric ancestry, and that either color of an asymmetric parent can appear in the symmetric form of an offspring.

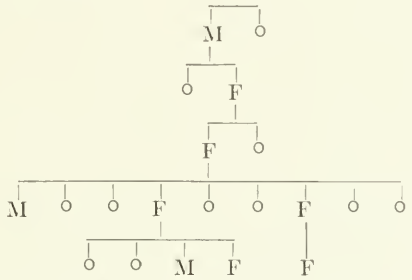

Fig. 1.—Gossage's case. M, affected male; F, affected female; ○, sex undetermined.

PARALYSIS OF THE CERVICAL SYMPATHETIC.

Within the past few years attention has been directed to the association in heterochromic eyes of alteration of a paretic nature of the cervical sympathetic. In fact, some observers, notably Bistis, are so enthusiastic over the close association that they have added to the classic Horner's syndrome (ptosis, myosis, anhidrosis, hemiatrophy facialis) heterochromia iridis as a symptom. Mayou[18] in 1910 was probably the first accurately to describe these changes, although Horner (quoted by Samelsohn[20]), as early as 1875, did draw attention to a case in which a light iris occurred on the same side with a ptosis of sympathetic origin. In

all only 33 cases have been reported,* 10 of which were complicated either with cataract or a slow uveitis.

From my own observation I can report four cases, the first two being of the same family, with positive symptoms of a sympathetic paralysis; the third gave less prominent symptoms, and the fourth presented only slight evidences of any sympathetic disturbance. My reason for here reporting case 5 along with the others is that we may assume that the heterochromia was due to certain trophic disturbances from a complete hemiplegia. Properly it belongs under the acquired classification if the history is correct.

Case 1.—Baby R., male, aged ten months, seen September 30, 1908; parents first noticed a drooping of the upper lid and a lighter color of the right eye when only a few weeks old. The father and mother are blonds and have blue eyes. The mother of the patient has a paternal aunt whose right eye is of lighter color; her brother has a drooping right upper lid with a lighter colored iris on the same side. This brother has a son (case 2) with the same abnormality on the right side. There is also another brother whose eyes are of the same color, and whose son has a drooping upper lid and light eye on the right side. This patient (baby R.) was born normally and has been healthy. The parents noticed that the child did not perspire on the right side of his brow.

The examination showed on the right side a decided ptosis, small pupil, light-blue iris, and a dryness of the skin of the right forehead as compared to the opposite side. As well as I could detect without a dilated pupil (the parents refusing eye drops), the fundus was normal and there were no corneal deposits. In July, 1915, nearly seven years later, I again examined the child. He had been well and healthy. The upper right lid still drooped, but not greatly, there was some suggestion of a facial atrophy on the same side, the pupil was still quite small compared to the left eye, yet it

* The following authors reporting: Galezowski,[11] 2 cases, 1 complicated; Horner's case, reported by Samelsohn;[20] Dethleffsen,[21] 5 cases, 1 complicated Lutz,[22] 13 cases, 3 complicated; Hutchinson,[2] 1 case; Alexander and Lander,[32] 5 cases, 2 complicated; Bistis,[39] 2 complicated cases; Mayou,[18] 3 cases, and Scalinci,[6] 1 case.

reacted well to light; the iris was blue, V. = 20/20, and fundus was normal, with no corneal deposits (without mydriasis). The left eye was normal and the iris brown (×192).

CASE 2.—Master D., aged five years. The family history is the same as case 1, the patient being his first cousin. The maternal grandmother gave me the history of the family and patient, and she stated that the condition to be described existed from birth. The patient's father (uncle to case 1) has blue eyes, and the mother's eyes are hazel, and I am told she is blind in one eye "from the rupture of a blood-vessel." At birth there was some abnormality (a breech presentation), although no instruments were used. There are two younger children with normal eyes of the same color.

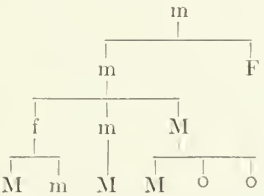

Fig. 2.—Author's case. M, affected male; F, affected female; m, normal male; f, normal female; o, sex undetermined.

Examination: Right, drooping upper lid, hemiatrophy facialis, palpebral fissure 5 mm., pupil active to light, 2 mm. in diameter. Color of iris was blue. Two drops of 1: 1000 solution of epinephrin did not dilate pupil. Cocain not used. Fundus normal; no corneal deposits. V. = 20/20. Left, dark-blue iris, with brownish pigmentation surrounding pupil, which measures 4 to 5 mm. Fundus normal. V. = 20/20.

The grandmother states that when the child is unwell, the drooping of the upper lid and the narrowing of the pupil of the right eye are perceptibly increased. Perspires on left side of face only (Fig. 2).

CASE 3.—J. L. M., male, aged fifty-seven years, states that all his life the left eye has been smaller than the right and of a lighter color. Family history negative; all children have dark eyes. The patient was brought to me as a re-

fractive case on account of headaches and dizziness. Patient is a brunette. Two years ago he had a stroke of apoplexy causing left hemiplegia, which now partially exists. He gives a positive luetic history, and has taken appropriate treatment, including salvarsan.

Examination: V. R. E. = 20/15 corrected. Palpebral aperture, 12 mm. Color of iris brown; pupil active to light and measures 2.5 mm. Many small vitreous opacities, but no corneal deposits. Typical retinal arteriosclerosis, *i. e.*, beaded and corkscrew arteries. Pupil does not dilate to epinephrin, and sluggishly to cocain, as compared to left. Tonometer, 20 mm. V. L. E. = 20/20 corrected. Palpebral aperture, 10 mm. Iris light gray and active to light. Pupil measures 3 mm., does not dilate to epinephrin, but actively and widely to cocain. No corneal deposits, but similar vitreous opacities and retinal changes as in right. Decided left hemiatrophy facialis and apparent enophthalmos. No anhidrosis. Tonometer, 20 mm. Hg. Visual fields contracted peripherally. Systolic blood-pressure 172.

CASE 4.—J. E. M., male, aged thirty-eight years. Family and hereditary history negative. Father's eyes brown, mother's blue (?). Patient is only member in family with different colored irides, and he states that he has had this difference all his life. He consulted me for pain in eyes after reading.

Examination: Right eye, H. + A. H. V. = 20/20. Pupil same size as left and reacted to light. Color of iris gray. Slight drooping of upper lid. Palpebral aperture 6 mm. Fundus normal. No corneal deposits.

Left eye: H. A. V. = 20/20; pupil active to light. Color of iris brown. Palpebral aperture, 8 mm. Decided bluish discoloration of sclera, arranged in patches above and out, and down and out. After using the same number of drops of homatropin in each eye for an hour to dilate the pupils for retinoscopy, it was observed that the right (light iris) was dilated to maximum with loss of accommodation, whereas the left pupil still reacted to light, with little loss of accommodation. Prolonged use of the drops did produce cycloplegia. Seen one month later. Slight ptosis on the right side, and right pupil 2.5 mm., whereas left 2 mm. No

response to epinephrin, and right pupil dilated to 4 mm. after twenty minutes from one drop of cocain. Left did not dilate. The only evidence of a sympathetic paralysis was a slight ptosis on the lighter side.

Case 5.—P. R. R., male, aged forty-three years, consulted me on account of defective sight of long duration in the right eye. Several members of his family have bad eyes, evidently refractive errors, and perhaps from ocular injuries. Blue or gray colored eyes predominate in the family. The patient is a decided brunette, with black hair, and states that when seven months of age he was paralyzed on the entire left side of body and face; that the left eye has always been of lighter color than the right. Is a farmer of moderate intellect. A general physical and neurologic examination was made by Dr. H. Crenshaw, who reported nothing abnormal except a general left hemiplegia. There were no positive evidences of a sympathetic paralysis. The eye examination is as follows: V. R. E. = light perception, extra-ocular muscles normal, pupil dilated and inactive, measures 5 mm. Iris light brown, with one area of intense brown pigment above and a sector-shaped area of gray below. Media clear. Typical cupping with atrophic changes of glaucoma simplex. Tension, 52 mm. Hg. Corneal anesthesia. V. L. E. = 20/30. Small pterygium. Pupil 2.5 mm., active, iris gray, slightly shallow anterior chamber, no drooping of upper lid. Fundus normal, cornea sensitive, no deposits. Field contracted peripherally, with an encroachment on nasal side. No enlargement of blind spot or scotoma. No anhidrosis, but there existed a decided asymmetry to the face; the left side much sunken, with a deepening of facial furrows. We assumed that the facial atrophy was due to the general hemiplegia and not to a seventh nerve involvement. It is difficult at this late day to explain in this case any direct connection between the light iris and facial hemiatrophy or hemiplegia on the left side, unless we can imagine that in infancy certain trophic disturbances developed as a result of the hemiplegia, but even this gives nothing very definite.

In closely reviewing the 33 cases reported, it is noticeable that in a good percentage the only evidence of a sympathetic

alteration was a narrowing of the pupil in the lighter eye. While this is evidence of a sympathetic paralysis, it is not altogether positive. In case 3, which I believe to be of sympathetic paralysis (having ptosis and hemiatrophy facialis), the pupil was larger and dilated under cocain more readily than its fellow. I take for granted that the authors have eliminated the many other influences which would cause a small pupil before positively declaring the case as one of sympathetic disturbance. In this connection it is interesting to note Pollock's[23]* well-known experiment, in which he caused a dilatation of the pupil from a mydriatic and a contraction by direct light in cases where he had completely excised the superior cervical sympathetic and the ciliary ganglia.

It should also be borne in mind that in rabbit experiments, where the superior cervical sympathetic ganglion has been completely removed, there is often a paradoxical dilatation of the pupil.†

Another fact to be noted, from the reported cases of heterochromia iridis associated with undoubted sympathetic paralysis, is that the paralysis was usually congenital or first noticed in early infancy, and the differences in the color of the irides developed at the natural time when changes take place in the color of the iris. In several cases (Mayou[19]) there was a history of forceps delivery, which was naturally supposed to be the cause of the paralysis. However, it is not difficult to find equally as many reports of a sympathetic

* Pollock concluded, after his experiments, that "evidence is given that a motor plexus with nerve-cells exists in the sphincter and in the dilator pupillæ of the rabbit's iris. This plexus lies between the individual cells of the muscles and contains fibers of extreme tenuity. The plexus persists after separation of the iris from the central nervous system. It may, therefore, be regarded in the same nature as the plexus of Auerbach and Meissner in the intestine."

† This phenomena has been observed by Langendorff,[24] where, several days after the ganglion has been removed, the pupil markedly dilates, and he explains it by vasomotor changes, *i. e.*, dilatation of the iris vessels causes the pupil to contract, while a narrowing produces a dilatation of the pupil. This vasomotor change also takes place in the ear: at first an increased warmth, and then later a coldness.

paralysis from forceps delivery without iris decoloration (Reese[25] and Burrows[26]), although in many of these reports no reference is made to the color of the iris. This lack of depigmentation in light-colored irides of blonds with an associated sympathetic paralysis can easily be explained. Mayou[27] has also mentioned cases of sympathetic paralysis without iris decloration associated with cervical rib. Of all the reported cases of sympathetic paralysis developing in adult life, Bistis[28] reports the only positive case where a change in the color of the iris subsequently developed; and the case is well worth reviewing.* Metzner[29] reports Mendel's case, in which there were certain suggestive peripheral depigmented changes in the iris.

A traumatic paralysis of the superior cervical ganglion or nerve in adult life is by no means an uncommon accident, and as yet no reports have been made of a subsequent iris decoloration. I personally have followed two such cases. I have also inquired of the older ophthalmic surgeons of large experience, who at one time advocated and practised sympathectomy for glaucoma, and in no instance has there been noted any subsequent change in the color of the iris.

Suggested Allied Causes.

It is noted that in case 3 there existed slight deafness, which, from the history and examination, suggests nerve impairment. I can offer no opinion from this limited experience as to whether there is any association between the auditory, ocular, and sympathetic disturbances. Mendel[30] mentioned

* Bistis' case. Female, aged thirty-eight years. The mother showed differences in the color of the irides, and the lighter eye showed cataract in an advanced age. For two years the patient's sight has failed, and the color of the right iris has changed. There was pain around the right eye, and facial hemiatrophy, together with anhidrosis of same side, appeared at same time as heterochromia. Author had seen patient often before onset of trouble for refraction, and the condition described did not exist. The eye showed ptosis, enophthalmus, descemetitis, and blue iris: pupil did not dilate as well to cocain as opposite side; no effect to epinephrin. Aqueous slightly turbid, tension subnormal, and lens changes.

a similar change in one of his cases. It is well known that deafness is often present in albino cats (Beaumont[31]), and in two cats which had heterochromia iridis and deafness Alexander and Lander[32] studied the anatomy of the eyes and ears. They found an absence of pigment in the perilymphatic cells of the auditory organ, and, on the other hand, the mesodermal pigment was absent in the lighter iris. These same authors called attention to the influence which certain gland-like structures at the posterior part of the ciliary body, first described by Collins,[33] might have in producing a depigmentation of the iris. According to Griffin,[34] these glands control the amount of pigment in the eye, shown by their absence in albino eyes and their imperfect development in feebly pigmented blue eyes. As plausible as this theory seems, the presence of these glands are nevertheless doubted by other able men (Alt[35] and Rutteman[36]), who were not even able to find their lumen. Finnoff[37] finds these so-called pigmented glands, but thinks that they are pigmented plugs, and, while he admits that gland-like bodies may be demonstrated, he considers the lumen an artefact, produced by the process of hardening, and a section of such tissue would resemble a tubular-like gland cut crosswise. Therefore, with this apparent uncertainty we could hardly accept at the present time the suggestion that the so-called ciliary glands of Collins have any influence in causing heterochromia iridis.

Experimental Work.

In regard to the experimental work, which has been done on animals by dividing the sympathetic nerve or excising the superior cervical ganglion, Angelucci[38] found that section of the ganglion produced trophic alterations of the eye, among them being a disappearance of the pigment of the iris, with an atrophy and depigmentation of the choroid, besides certain well-defined alterations of the blood-vessels of the iris. Bistis[39]

produced similar changes in the iris, and in one case he was able to demonstrate a cloudiness of the aqueous, and found in sections deposits on the posterior surface of the cornea which, to his mind, was highly suggestive of an inflammatory process. Later, Metzner[29] and Wolfflin added evidence that a sympathetic paralysis caused a distinct loss of pigment in the iris, and also recorded other changes which had previously been observed, such as epiphora, flattening of the cornea (Angelucci), changes in the motility of the nictitating membrane, and hypotonia. This latter observation was to a certain extent agreed with in our experiments.

By more recent experiments on rabbits these same authors concluded that the superior sympathetic ganglion must have an independent tonus as regards the dilatation of the pupil, for if on one side division of the nerve was made below the ganglion, and on the opposite side the ganglion was extirpated, the pupil was smaller on the extirpated side. Their experiments also showed that the extirpation of the ganglion was never accompanied by a depigmentation of the iris, but that a resection of the nerve below the ganglion was followed by a decoloration of the iris.

These last-mentioned experiments present new phases to the subject with which we have had no experience, and which are quite contrary to the general accepted physiology of the sympathetic system.

In this connection it is of interest to know that Galezowsky reported a case of heterochromia where, in the lighter eye, the pupil and the palpebral fissure were wider, and the eye was more prominent, than its fellow. Obviously, there were symptoms of a sympathetic stimulation or irritation.

THE AUTHOR'S EXPERIMENTS.

An Excision of the Cervical Sympathetic Ganglion in the Rabbit.—Guinea-pigs were first employed in our experiments, mainly to perfect the operative technic of the re-

moval of the superior cervical ganglion, and later Belgian hares were used. After the operation the ganglion was preserved in normal saline solution, later stained with methylene-blue (vital stain) and examined microscopically.

Light ether anesthesia was administered, the fur on the under surface of the neck was shaved, the part washed with soap and water, sponged with alcohol, and then painted with tincture of iodin. The ganglion was rapidly exposed and grasped with a pair of tissue forceps and excised with scissors. The skin incision was closed with continuous silk sutures, and a sterile gauze dressing applied with adhesive tape. Apparently the animals did not suffer in any way from the operation, and were quite alert shortly afterward. There were no infections.

As the rabbit's pupil is often oval (the vertical diameter being the greatest), pupillary measurements were made with a millimeter rule along the horizontal meridian of the cornea —that is, from canthus to canthus. The eyes were exposed to daylight from a northernly direction. Tonometric readings were made with the Schiötz instrument, and it was applied the moment after an instillation of one drop of a 4 per cent. aqueous solution of cocain. The lids were gently retracted with the fingers, and usually the readings were easily and presumably accurately made, although at times the animals were exceedingly nervous and frightened, which apparently accounted for the wide variations in the intraocular tension. The 7.5 mg. weight was employed, but when there was doubt of the accuracy, other weights were used as controls and the readings usually corresponded.

It was exceedingly difficult to distinguish early decoloration of the irides, and it is possible that one's imagination had its influence in causing certain discrepancies made in our early observations, but when a decided change was noted, this opinion was concurred in by three or four other observers. Eyes which showed no great change in the color

The above chart represents the intraocular tension and pupillary measurements of the rabbits used in the experiments. The first readings indicate the tension and size of the pupil of the right eye of each rabbit before operation, while all other readings were made after operation. The letters indicate the following: E, epinephrin; C, cocain; O, operation, and D, death.

during life presented a marked difference after enucleations, and when held side by side for a closer comparison.

Five guinea-pigs were used, and the four that survived all showed a narrowing of the palpebral aperture and myosis, but no macroscopic change in the color of the iris on the operative side even seven months after the operation. Weeping of the eye on the operative side was noticed as a symptom in three cases after recovery from ether anesthesia, which later disappeared.

Rabbit No. 1.—November 18, 1915: Male, weight 1620 gm. Color of irides light brown. Size of right pupil 6.5 mm.; contracts to light. Left, 6 mm., contracts to light. Tension: Right, 22 mm. Hg; left, 27 mm. Hg. Fundus normal.

Operation: Removal of right superior cervical sympathetic ganglion. Right pupil immediately contracted, and grows smaller; left dilated and does not respond to light. Lacrimation right eye.

November 19th: Right pupil smaller than left.

November 22d: Pupils of equal size; reaction to light—right more sluggish. Palpebral aperture same size.

November 27th: Right pupil smaller than left.

November 30th: Right pupil 6 mm.; left, 8 mm.

December 2d: Right pupil 6.5 mm.; left, 8 mm. Nictitating membrane not affected. Tension: Right, 27 mm. Hg; left, 26 mm. Hg. Right palpebral aperture narrower than left. No increased lacrimation. Color of iris same. Fundus normal.

December 21st: Pupils: Right, 6.5 mm.; left, 7 mm. Tension: Right, 27 mm. Hg; left, 29 mm. Hg. Right iris appears to be of lighter color than left.

December 29th: Narrowing of right palpebral aperture. Pupils: Right, 7 mm.; left, 8 mm. Tension: Right, 27 mm. Hg; left, 24 mm. Hg.

January 20, 1916: Slight ptosis, right side. Pupils: Right, 6 mm.; left, 7 mm. Tension: Right, 27 mm. Hg; left, 22 mm. Hg. The iris has in the upper part a "washed-out" appearance.

February 3d: Slight ptosis, right side; pupils equal,

5 mm. Fundus normal. Tension: Right and left, 13.5 mm. Hg.

February 7th: Rabbit died of unknown cause. Pupils: Right, 4 mm.; left, 6 mm. Eyes enucleated. Color of right iris distinctly lighter than left. (See description under histopathology.)

Rabbit No. 2.—December 2, 1915: Male, weight 2310 gm. Irides dark brown. Pupils 7 mm. in horizontal diameter. Tension each eye, 26 mm. Hg.

Operation: Removal of right superior cervical sympathetic ganglion. Rabbit did not react from anesthetic.

Rabbit No. 3.—December 7, 1915: Female; weight 1220 gm. Irides dark brown. Pupils, 7 mm. Tension: Right and left, 26.5 mm. Hg. Fundus normal.

Operation: Removal of right superior cervical sympathetic ganglion. Right pupil contracts to 5 mm. and both react promptly to light. No weeping or ptosis.

December 8th: Pupils: Right, 5 mm.; left, 6 mm.; slight ptosis.

December 14: Pupils: Right, 6 mm.; left, 7 mm.; slight ptosis. Nictitating membrane shows slightly on right and not at all on left.

December 21st: Pupils: Right, 6 mm.; left, 8 mm. Tension: Right, 22 mm. Hg; left, 26.5 mm. Hg. No change in color of iris. Fundus normal.

December 28th: Pupils: Right, 6.5 mm.; left, 7 mm.; right ptosis. Fundus normal. Tension: Right, 22 mm. Hg; left, 26.5 mm. Hg.

January 20, 1916: Pupils: 6 mm.; right ptosis. Tension: Right, 42 mm. Hg; left, 18.5 mm. Hg. No change in color of iris. Fundus normal.

January 31st: Died. No change in size or color of iris. Eyes enucleated.

Rabbit No. 4.—December 8, 1915: Male; weight, 1530 gm. Color of irides brown. Pupils, 8 mm. Fundus normal. Tension, each eye, 26 mm. Hg.

Operation: Removal of right superior cervical sympathetic ganglion. Immediate contraction of right pupil—right, 6 mm.; left, 8 mm.

December 9th: Pupils: Right, 7 mm.; left, 9 mm.; ptosis right; nictitating membrane not visible on either side.

December 13th: Pupils: Right, 6 mm.; left, 8 mm.

December 21st: Pupils: Right, 6 mm.; left, 7.5 mm. Tension: Right, 24 mm. Hg; left, 26.5 mm. Hg. Fundus normal. Right ptosis; lighter color to right iris.

December 29th: Pupils: Right, 7 mm.; left, 8 mm. Tension: Right, 18.5 mm. Hg; left, 26.5 mm. Hg. Fundus normal. Right ptosis.

January 20, 1916: Pupils: Right, 6 mm.; left, 7 mm. Right ptosis. Fundus normal. No apparent change in color of iris. Tension, each eye, 22 mm. Hg.

February 3d: Pupils: Right, 6.5 mm.; left, 8 mm. Right ptosis. Tension: Right, 16 mm. Hg; left, 18.5 mm. Hg.

February 24th: Same as above. No change in color of iris. Fundus normal.

March 2d: Pupils: Right, 6 mm.; left, 7 mm. One per cent. aqueous solution homatropin in each eye; after fifteen minutes in dark room, right, 8 mm.; left, 9 mm.

March 16th: Pupils: Right, 7 mm.; left, 8 mm. One drop 1:1000 epinephrin solution in each eye; after fifteen minutes in dark room, right pupil, 10 mm.; left, 9 mm.

March 23d: Pupils, 7 mm. Slight right ptosis. Tension, each eye, 18.5 mm. Hg. Two drops 4 per cent. aqueous solution cocain in each eye; after fifteen minutes in dark room, pupils 8 mm.

April 13th: Pupils: Right, 7 mm.; left, 8 mm. Tension: Right, 24 mm. Hg; left, 31 mm. Hg. Right ptosis. Fundus normal. Slight discoloration of right iris. One drop 4 per cent. solution cocain in each eye; after fifteen minutes in dark room, pupils, right, 7 mm.; left, 8 mm.

May 4th: Pupils: Right, 6.5 mm.; left, 8 mm. One drop 4 per cent. cocain solution in each eye; after twenty-five minutes, right pupil, 7 mm.; left, 8.5 mm. One per cent. homatropin solution in each eye; after twenty minutes, left pupil dilates more than right. No fundus change.

May 25th: Pupils: Right, 7 mm.; left, 8.5 mm. The right iris is slightly lighter than the left. Tension: Right, 18 mm. Hg; left, 30 mm. Hg. Aqueous in right eye appears turbid, but there are no corneal deposits. Attached to the

under surface of the right iris, or rather hanging from it at twelve o'clock, is a small black tag which moves with the iris excursions. In the horizontal diameter of the cornea, near the limbus of the temporal side and within the cornea proper, is a refractile body.

June 22d: Pupils: Right, 6.5 mm.; left, 8 mm. The difference in the color of the irides does not appear to be so marked. The aqueous in the right eye is not so turbid as at the previous examination, and the iris tag is not present. Homatropin dilated both pupils evenly. The general view of the fundus of the right eye does not appear to be as clear as the left. After mydriasis the iris tag is seen, but it is not so large.

July 6th: Very slight difference in the color of the irides. There is a second pigment tag of the iris at about 1 o'clock.

July 27th: Pupils: Right, 7 mm.; left, 8 mm. Very slight difference in the color of the irides, but one pigment tag hanging from the iris, and that at twelve o'clock. There are two distinct opacities seen in the posterior portion of the lens. Epinephrin solution 1: 1000 dilates the right pupil to 11 mm. but the left remains the same.

August 10th: Opacities in lens still present. It has been noticed for some time that it is more difficult to see the fundus in the right than in the left eye, due to detritus on cornea. It was also noticed that after the instillation of epinephrin the cornea became clearer.

August 24th: There is a slight difference in the color of the irides.

October 19th: Pupils: Right, 7 mm.; left, 8 mm. There is only a slight difference in the pigmentation of the two irides. The pigment tag hanging from the iris is still present. The opacities in the lens noted at previous examinations are not seen.

January 4, 1917: Rabbit in good health. Pupils: Right, 6.5 mm.; left, 8 mm. There is a decided difference in the pigmentation of the two irides. No opacities seen in the lens or vitreous. Iris tag still present.

January 29th: Rabbit died of unknown cause. Eyes enucleated for examination.

Rabbit No. 5.—December 9, 1916: Male, two months old:

weight, 810 gm. Color of irides, brown. Pupils: 6 mm. Fundus normal. Tension (obtained with difficulty, as animal was nervous): right and left, 26 mm.

Operation: Removal of right superior cervical sympathetic ganglion. Immediately right pupil contracts to 4 mm. Nictitating membrane invisible.

December 9th: Pupils: Right, 6 mm.; left, 8 mm. Right ptosis.

December 21st: Pupils: Right, 4.5 mm.; left, 6 mm. Tension: Right, 17.5 mm. Hg; left, 27.5 mm. Hg.

December 29th: Pupils: Right, 5 mm.; left, 6 mm. Right ptosis. Tension: Right, 26.5 mm. Hg; left, 22 mm. Hg.

January 18, 1916: Died. Pupils of equal size. Eyes removed (hemorrhage in right orbit); when held together the right iris is paler than left, and the decrease of the pigment seems to be more marked toward the pupillary margin. (See description under histopathology.)

Rabbit No. 6.—December 9, 1915: Male, two months old; weight, 800 gm. Color of irides, brown. Pupils, 6.5 mm. Fundus normal. Tension (obtained with difficulty as animal was nervous): Right, 27.5 mm. Hg; left, 28 mm. Hg.

Operation: Removal of right superior cervical sympathetic ganglion. Pupil on right side, 6 mm.; left, 8 mm. Nictitating membrane not visible.

December 10th: Pupils: Right, 5.5 mm.; left, 6.5 mm. Right ptosis.

December 21st: Pupils: Both, 6.5 mm. Tension: Right, 17.5 mm. Hg; left, 26.5 mm. Hg. Slight right ptosis.

December 29th: Pupils: Right, 6 mm.; left, 7 mm. Right ptosis. No change in color of iris. Tension, both eyes, 26.5 mm. Hg.

January 13, 1916: Died. Pupils: Right, 4 mm.; left, 8 mm. Eyes removed (extensive hemorrhage in bottom of orbits). Right iris distinctly paler than left.

Rabbit No. 7.—December 14, 1915: Male; weight, 2800 gm. Brown irides. Pupils, 7 mm. Fundus normal. Tension, each eye, 21 mm. Hg.

Operation: Removal of right superior cervical sympathetic ganglion. Right pupil immediately contracted to 6 mm.; left, 9 mm. (cocain).

December 21st: Pupils: Right, 7 mm.; left, 8 mm. No difference in palpebral aperture. Tension: Right, 9.5 mm. Hg; left, 14 mm. Hg.

December 29th: Pupils, 7.5 mm. Slight ptosis in right side. Fundus normal. Tension: Right, 16 mm. Hg; left, 18.5 mm. Hg.

January 20, 1916: Pupils, 8 mm. Tension: Right, 11.5 mm. Hg; left, 18.5 mm. Hg. Right iris slightly paler than left, especially in upper posterior quadrant.

February 3d: Animal apparently paralyzed in hind legs. Pupils, 8 mm. Fundus normal. Slight ptosis right side. Tension: Right, 11.5 mm. Hg; left, 9.5 mm. Hg. Right iris slightly lighter than left.

February 5th: Died.

Rabbit No. 8.—December 14, 1915: Male; weight, 2620 gm. Brown irides. Pupils, 8 mm. Fundus normal. Tension (obtained with great difficulty as animal was frightened and nervous): Many readings of the tonometer, using different weights to check results; right, 7 mm.; left, 8 mm. Hg.

Operation: Removal of right superior cervical sympathetic ganglion. Right pupil contracts to 7 mm.; left, 10 mm. (cocain).

December 21st: Pupils: Right, 7 mm.; left, 6 mm. Slight ptosis right side. Tension (with difficulty): Right, 26 mm. Hg; left, 22.5 mm. Hg.

December 29th: Pupils, 8 mm. No difference in palpebral apertures. Tension (animal quiet): Right, 12.5 mm. Hg; left, 26.5 mm. Hg. Right iris slightly lighter in color than left.

January 20, 1916: Pupils: Right, 5.5 mm.; left, 7 mm. Slight ptosis right. Right iris slightly lighter than left, especially in upper temporal quadrant. No change in fundus. Tension: Right, 11.5 mm. Hg; left, 23.5 mm. Hg.

February 3d: Pupils: Right, 6.5 mm.; left, 7.5 mm. Tension: Right, 9.5 mm. Hg; left, 12.5 mm. Hg.

February 23d: Pupils, 7 mm. Tension, each eye, 26.5 mm. Hg. Right iris lighter than left.

March 2d: Pupils: Right, 6 mm.; left, 7 mm. One drop aqueous solution homatropin in each eye. Animal placed

in dark room, and after fifteen minutes pupils dilated equally to 9 mm.

March 16th: Pupils: Right, 7 mm.; left, 9 mm. One drop 1: 1000 epinephrin solution in each eye, and after fifteen minutes in dark room, right pupil, 13 mm.; left, 9 mm.

March 23d: Pupils: Right, 7 mm.; left, 8 mm. Tension, each eye, 5 mm. Hg. Right iris of lighter color than left. Two drops of 4 per cent. aqueous solution cocain in each eye, and after twenty minutes in dark room pupils did not dilate, *i. e.*, right, 7 mm.; left, 8 mm.

April 13th: Pupils: Right, 7 mm.; left, 8 mm. Slight ptosis right. Tension: Right, 31 mm. Hg; left, 31 mm. Hg. Right iris distinctly paler than left. A drop of 4 per cent. cocain solution in each eye, and after twenty minutes both pupils measured 8 mm.

May 4th: Pupils measure 7.5 mm. After twenty-five minutes use of 4 per cent. cocain solution in each eye; right pupil measures 7.5 mm.; left, 9 mm. The right iris is distinctly paler than the left. A drop of 1 per cent. homatropin solution was instilled in each eye, and after twenty minutes the left pupil dilated wider than the right. There was no appreciable fundus change.

May 25th: Pupils: Right, 7 mm.; left, 8 mm. Tension: Right, 11 mm. Hg; left, 32.5 mm. Hg.

June 22d: Pupils, 7 mm. Tension: Right, 18 mm. Hg; left, 12 mm. Hg.

July 6th: Right iris still lighter in color than left.

July 27th: Right pupil smaller than left, and color of right iris lighter than left. No fundus change. Animal killed by blow on back of neck. Eyes enucleated and placed in 10 per cent. formalin solution. (See description under histopathology.)

Rabbit No. 9.—December 16, 1916: Female; weight, 2600 gm. Brown irides. Pupils, 7 mm. Fundus normal. Tension: Right, 16 mm. Hg; left, 17 mm. Hg.

Operation: Removal right superior cervical sympathetic ganglion. Right pupil contracts to 6.6 mm.; left, 8 mm.

December 21st: Right pupil, 6 mm.; left, 7.5 mm. Slight ptosis right. Tension: Right, 11 mm. Hg; left, 18.5 mm.

December 29th: Slight ptosis right. Pupils: Right, 7 mm.; left, 8 mm. Fundus normal. Tension: Right, 14 mm. Hg; left, 17.5 mm. Hg.

January 20, 1916: Pupils: Right, 6 mm.; left, 7 mm. Slight ptosis right. Tension: Right, 11.5 mm. Hg; left, 20 mm. Hg. Right iris generally paler than left.

February 3d: Pupils: Right, 6 mm.; left, 7 mm. Slight ptosis right. Tension: Right, 17.5 mm. Hg; left, 17 mm. Hg.

February 23d: Slight ptosis right. Pupils: Right, 6 mm.; left, 7 mm. No change in fundus. Tension: Right, 16 mm. Hg; left, 18.5 mm. Hg.

February 27th: Died in labor. Pupils: Right, 3 mm.; left, 8 mm. Eyes enucleated. Right iris distinctly paler than left.

TABLE 1.—TONOMETRIC READINGS OF THE RIGHT EYE BEFORE AND AFTER OPERATION.

Rabbit	Mm. Hg		Mm. Hg
No. 1	Average tension before operation	22	Total Average after 24
No. 3	Average tension before operation	27	Total Average after 22
No. 4	Average tension before operation	26	Total Average after 21
No. 5	Average tension before operation	26	Total Average after 14.8
No. 6	Average tension before operation	27.5	Total Average after 22
No. 7	Average tension before operation	21	Total Average after 12.1
No. 8	Average tension before operation	7	Total Average after 17.8
No. 9	Average tension before operation	16	Total Average after 14.3
General average tension before operation,		21.5	after operation 18.5

A summary of the above experiments on the nine rabbits, after a complete excision of the right superior cervical sympathetic ganglion, shows:

1. No marked influence on lacrimation. There was an increase in one case immediately after operation; the observation made August 10th on No. 4 was probably due to a disturbance of lacrimation.

2. The nictitating membrane was not affected by the operation.

3. A paresis of Müller's muscle, which caused drooping of the upper lid in all cases on the operated side.

4. The average horizontal diameter of the pupil before

operation was 7.6 mm. It contracted immediately after the operation, and usually so remained, the average diameter then being 6 mm. In those cases where epinephrin was instilled into the conjunctival sac it caused a dilatation of the pupil on the operated side.

5. There was no appreciable ophthalmoscopic change in the retina and choroid. In rabbit No. 4, as seen from the notes, there were certain interesting developments in the iris which I was not able to explain. The tags of pigment undoubtedly developed from the epithelial layer, but what influence the operation had in causing them is a matter of conjecture. While I could not detect deposits on the posterior surface of the cornea, a cloudiness of the aqueous was surely present at one time; also there were opacities in the media, which I placed in the posterior part of the lens, as the rabbit's lens is very thick. These opacities later disappeared.

6. There was an appreciable gross change in the color of the iris on the operated side in all cases except one (rabbit No. 3 died twenty-four days after operation). The longer the animal lived, usually the more distinct was this decoloration, and where the eyes were enucleated and placed side by side for closer comparison, the change was more decided. About five weeks after operation changes in the color of the iris were first detected, although in one case (rabbit No. 8) a suspected change took place in fifteen days, which was more positive during the fifth week. Unquestionable variations in the color of the iris were noted in rabbit No. 4; that is to say, one time the change was barely perceptible, and at other times the decoloration was decided.

7. The intraocular tension (see chart and table) shows varying estimates, without any uniformity. Taking an average first reading before operation, the right eye was 21.5 mm. Hg and left, 22 mm. Hg. A combined average of all readings after operation (omitting the first reading)

on the right was 18.5 mm. Hg, and the average of all readings on the left was 21.2 mm. Hg. This observation would lead us to believe that the removal of the superior cervical sympathetic ganglion lowers the intraocular tension in the eye on the operative side.

8. The age or weight of the animal apparently had no influence in these experiments.

Cases Complicated by Cataract or Uveitis.

There is another classification of primary heterochromia iridis to be considered, namely, those cases in which there is an uveal affection, evidenced by deposits on the posterior surface of the cornea, which may and usually do terminate in cataract. These complicated cases may or may not be associated with the apparent disturbances of the sympathetic. Of the 33 cases of iris decoloration associated with sympathetic paralysis, 10 cases were complicated with uveitis or cataract.

Lawrence and Hutchinson were among the first to describe the association of cataract with heterochromia iridis, and Fuchs[41] claims priority for first calling attention to the fact that chronic cyclitis with deposits occurred in the lighter eye. He also called attention to the fact that in his series of 38 cases, 20 occurred within the first three decades, 9 in the fourth decade, and the remaining number scattered. It was his opinion that heterochromia was usually due to some anatomic difference in the stroma of the iris, and when the retinal layer was affected (which condition he had observed), it was then due to a chronic inflammation. He often noted that the pupil was larger on the lighter side.

Butler[8] regards the cause as unknown, but when associated with a sympathetic paralysis, the chronic cyclitis may be comparable to the iritis and iridocyclitis which are occasionally seen in herpes ophthalmia, and are usually regarded

as trophic disturbances. The same author also describes a train of symptoms which in part are as follows:

The change in the color of the iris may last several years before other complications set in, but sooner or later the patient complains of bad sight in the blue eye. There is never pain or redness, possibly a ciliary blush. There is no associated dyscrasia, although patients look "out of sorts." Occasionally there is a transient albuminuria. The eye shows fine keratitis punctata, which soon covers the whole posterior surface of the cornea, although in other cases it is arranged sector shape. An exudation may develop in the vitreous, followed by opacities of the lens, which are often punctate, and glaucoma is not an infrequent complication. Choroiditis and optic neuritis have been observed. The prognosis is generally bad. The disease usually begins in youth, and is slow and may extend over a period of years or even decades.

Contrary to the views of Butler and other observers, Galezowski is of the opinion that the depigmentation is preceded by a chronic inflammatory condition. On the other hand, Malgat regards the heterochromia as due to a difference in the blood circulating in the iris or ciliary body, and from a lack of nourishment to the lens, a cataract being the natural consequence. Hirschberg is of this same opinion, but thinks that the defective circulation starts *en embryo* or before the development of the iris pigment. This condition may remain stationary for years, to again become active and cause a uveitis and cataract. Gunn refers to the iris which contains sectors of different colors, and which subsequently regains its color, and he thinks that it is suggestive of an abnormal enervation. Finally Dor thinks that, besides the congenital heterochromia (he does not mention sympathetic paralysis), there exists a disease of the eye yet unknown, which begins in heterochromia and ends in cataract.

It is natural to ascribe the formation of cataract in these cases directly to the absence of iris pigment, and also to the influence of the action of ordinary daylight. But Sym has drawn attention to the fact that albinos are no more liable to cataracts in proportion to their number than are the normally pigmented.

Others attribute the cataracts to the chronic cyclitis which frequently accompanies heterochromia, and, as certain other intraocular changes are frequently noted, and as occasionally serious complications follow extractions, it is a commonly accepted cause.

Scalinci regards the cataract as due to changed physiochemical relations with the circulating nutritive fluids, or to biologic alterations in the content of the nutritive fluid itself, due to vasomotor paralysis.

Knapp, discussing Ellett's[46] paper on "Heterochromia Iridis, Heterochromic Cyclitis, etc.," was of the opinion that disturbances of the sympathetic system had very little to do in producing these changes (cyclitis). In practically all his cases the patients have reacted to the tuberculin test, and in a large number they have done well with tuberculin therapy.

HISTOPATHOLOGY OF THE IRIS.

A brief review of the histology of the iris may be helpful to those (as it was to the author) who study the microscopic changes in an heterochromic iris. Brown's[44] translation of Salzmann's "Anatomy of the Eye" is my authority for the following description:

The arrangement of the layers of the iris is regular in the middle part of the ciliary zone. It consists—(1) of an endothelial layer, which is difficult of histologic demonstration, continuous from the inner layer of the cornea. It lies in intimate contact with (2) the anterior border layer, made up principally of cells, which in reality is only a modification of the iris stroma. The cells are chromophores, like

the stroma cells, and usually possess two or three processes. It gives color to the iris, not alone through its pigment content, but also in its density; blue irides have a delicate border layer and almost unpigmented cells, while brown irides have a thick border layer and heavy pigmented cells. A complete absence of pigment in the border layer and stroma probably occurs in the new born or in very young children. These, therefore, at times actually have blue eyes, for the blue color is due only to the fact that a clouded but colorless medium (border layer and stroma) lies in front of a dark background (pigment epithelium). This difference also comes out when color in different sectors of the iris varies. Next (3) is the vessel layer which forms the main mass of the iris. It contains the blood-vessels and nerve plexuses, held together by a loose delicate stroma. The blood-vessels enter the iris root in large bundles and branch into finer branches and pass through the ciliary zone in a radial direction in several layers. The vessels show a corkscrew arrangement to adapt themselves to the changing contraction of the iris tissue. In the pupillary zone the course is altered by the formation of circles from anastomosis.

The vessels of the iris are characterized by a thick adventitia consisting of a finely fibrillated collagenous tissue, which often exceeds the diameter of the vessel lumen. The arteries have a thin muscularis and a weak intima, and the veins have perivascular sheaths bordering immediately upon the endothelium.

The nerves of the iris likewise advance through the iris root in large trunks, and build a plexus in front of the larger vessels. Nerves are practically not to be demonstrated in cut sections. The nerve-fibers end partly in the stroma (sensory fibers), partly in the vessels (sympathetic fibers), partly in the sphincter pupillæ, and partly in the dilator (motor fibers).

The spaces between the blood-vessels and the nerves are filled by the iris stroma proper, which is a loose collagenous tissue consisting of discrete fibrillæ containing pigment stroma cells (chromatophores), non-pigmented stroma cells, and clump cells. The chromatophores are grouped principally about the vessels and nerves, and each cell shows a small oval body which stains well and an oval nucleus not sur-

rounded by pigment; the processes are slender and long, and unite into a plexus with those of their neighbors. Other pigment-cells are also found which are termed clump cells; they are large and more densely pigmented, and are found in the neighborhood of the sphincter pupillæ.

The back surface of the iris is termed the ectodermal layer, and it is a continuation forward of the pars ciliaris retinæ. It is divided into an outer leaf (the dilator pupillæ) and the inner leaf (the pigment epithelium). The dilator pupillæ, like the sphincter, is an epithelial muscle or a "myoid plate," as it is called, *i. e.*, its fibers develop out of epithelial cells, and while a complete transition from an epithelial cell into a muscular cell occurs in the sphincter pupillæ, this takes place in only a part of the cell in the dilator pupillæ. When typically developed, it consists of a spindle-cell formation with an oval nucleus and a moderately pigmented protoplasm, extended at each end into an unpigmented fiber layer process. From the position of these cells two layers are recognized—one a posterior border layer and then the layer of pigmented spindle-cells.

This pigmented muscle or myoid plate extends along the posterior surface of the vessel layer of the iris, from the ciliary border of the sphincter pupillæ almost to the root of the iris, and at the sphincter zone the elements of the dilator are united to those of the sphincter.

The inner leaf or pigment layer has an epithelial character throughout, and is a direct continuation of the tunica interna of the ciliary body. Its cells are so densely filled with dark-brown pigment granules that cell borders or nuclei are not visible, except in bleached preparations.

But few writers have attempted to explain the alterations in the iris associated with a sympathetic paralysis; or, in other words, how a sympathetic paralysis caused an iris decoloration. The investigation of Angelucci on rabbits and dogs offers us a reasonable explanation. He concluded that the eye resents trophic disturbances on account of the influence which the cervical sympathetic exerts on the movements of the walls of the blood-vessels (vasomotor effect). It is well known that for a time after the superior sympathetic

has been excised there is a dilatation of the blood-vessels, which in the case of the rabbit can easily be demonstrated in the ears. Later it is followed by a thickening of the vessel-wall which tends to contract the lumen. With the weakening of the nutritive changes thus brought about there is an arrest of development of the eye, and in the case of the iris there is a more or less pronounced degree of simple atrophy, with its accompanying decoloration.

I cannot find recorded a report of a microscopic examination of a human decolored iris associated with sympathetic paralysis. Bistis, in his examination of rabbit eyes, found a great reduction in the pigment and a thickening of the vessel-wall. The pigment in the anterior layer was much reduced and appeared in small aggregation. The iris stroma possessed no pigment cells, but contained a large number of nuclei which occupy the place of the stroma cells. These nuclei are round or oval, and are situated in a basement substance composed of fibrillary connective tissue. He was also able to detect deposits on the posterior surface of the cornea. These findings, together with a cloudiness of the aqueous and the vessel-wall changes, he thinks are suggestive of an inflammatory condition.

Fuchs was the first to examine a heterochromic iris occurring in cataracts. In one case he could demonstrate cells on the back of the cornea. There was some depigmentation in the anterior layer, principally of small pigment granules. In the deeper layer there were only isolated remains of the branching pigment stroma cells, and these had lost their shape and had become round. These should not be confounded with clump cells found near the sphincter and Bruch's membrane. The retinal pigment layer was normal. The texture of the stroma had entirely changed, and the network of the branching stroma cells had been replaced by nucleated cells of a different character. There was no accumulation of nuclei around the blood-vessels, as is seen in

an inflammatory infiltration of the iris. Protoplasm was scanty and occurred around the nucleus like a seal ring. Lymphocytes were only occasionally found, and no polynuclear leukocytes were seen. The blood-vessels showed a hyaline degeneration; the nuclei were scarce, as were the endothelial nuclei. Complete closure of the lumen was observed.

Lagleyze[45] confirmed Fuchs' observations, except there were greater atrophy and depigmentation in the anterior layers.

AUTHOR'S FINDINGS.

Rabbit No. 1.—Interval of eleven weeks and two days between operation and death. The notes show that after enucleation the right iris was "distinctly lighter" than the left, although a suggestion of discoloration was noted some time before death. Microscopic study of each eye did not show any decided variation from the normal, except in a few of the thinnest sections there was noted an appreciable difference in the general pigmentation in the extreme anterior border layer.

Rabbit No. 5.—Interval of five weeks and four days between operation and death. After operation the right iris appeared lighter than the left, but no difference was detected microscopically.

Rabbit No. 8.—Interval of approximately twenty-eight weeks between operation and death. There was noted before and after enucleation a decided difference in the pigmentation of the irides. Our findings did not differ materially from those of Bistis, Fuchs, and Lagleyze. The greatest changes were noted in the anterior border layer, where there was a general atrophy of the branching pigment cells; this atrophy was likewise present to a large extent in the chromophores of the vessel layer. In certain areas along the margin of the anterior border layer there appeared to be a disorganization or absorption of the pigment cells, which probably corresponded to the lighter areas seen on the iris during life. The pigment epithelium was unaffected, and there was no sign of inflammation. There was a difference in the

thickening of the vessel-walls, due to a hyaline degeneration, although all the iris vessels were characterized by a thickened adventitia. We could not detect the marked vessel changes mentioned by other observers, which was probably due to the short interval between the operation and enucleation in this particular animal, as compared to Fuchs' examination of the human heterochromic eye.

Rabbit No. 4.—Interval of thirteen months and twelve days between the operation and death. Before operation there was a noticeable difference in the color of the irides. With the low magnification the general comparison of the sections showed a marked difference. In the section of the right eye there was a "washed-out" appearance in the anterior border and vessel layer, with a noticeable depigmentation of the cells surrounding the larger vessels. These changes were especially noted in the middle and upper thirds of the iris, and to some extent in the ciliary region.

The sections did not reveal any cause for the changes detected during life (*i. e.*, the iris tags and lens opacities (?) described in the notes).

With the higher magnification, the changes were similar to those described in rabbit No. 8, except for an increase in adventitia thickening.

Acquired Secondary Heterochromia Iridum.

Cases of heterochromia iridum occurring secondary to some acquired intraocular state are common to all ophthalmologists of any experience. A severe iridocyclitis, foreign bodies (metal) in the globe, degenerative changes of myopia, and chronic glaucoma are the common causes.

Comment.

Those who have studied the literature of heterochromia iridis, or even casually glanced over the many reports and discussions which I have very briefly collected, must have been impressed with the vastness of the subject and the lack of unity of opinion of the many authors. I, therefore, with trepidation hesitate to express an opinion, unless substan-

tiated by facts, and unfortunately in this character of investigation "cold" positive facts are very few. I shall then confine myself to a systematic arrangement of the cases and causes of heterochromia, and express my personal opinion only where experience warrants it. I do not feel, however, that the last word on heterochromia iridis has been written, yet we do know a great deal concerning it.

I firmly believe in the influence of heredity, and with the subject under consideration I am convinced that in a proportionate number of these cases certain tendencies are inherited which cause a difference in the color of the irides. This statement especially applies to the cases in which there is not even the suggestion of any alteration of the cervical sympathetic system. In this country, where our people are not only a mixed hue, but of many nationalities, a proportion of our fairly numerous cases of heterochromia are not all pathologic specimens, but simply "freaks" of nature.

Regarding the inheritance of a sympathetic paralysis, I believe that its cause may be transmitted, and its manner of producing heterochromia is similar to any other form of congenital sympathetic paralysis, including those of traumatic origin (forceps deliveries). Assuming then this sympathetic disturbance in fetal life, it would not be difficult to explain the arrested development of the iris pigment on the involved side, and we could suppose the same changes in those cases occurring in early life where the iris pigment has developed. These changes can be explained only by nutritional disturbances of trophic origin—first, a vasodilatation, then a hyaline degeneration of the adventitia, and finally a contraction of the vessel lumen.

The great number of clinical reports show the difference in the color of the irides to have taken place within the first few months or years of life, when the pigment cells were young and apparently easily absorbed; on the other hand, I have not found a record where a sympathetic paralysis

in the adult, produced by trauma or operation, ever caused iris depigmentation.

A chronic uveitis (deposits on the posterior corneal surface) is often recognized in association with heterochromia with or without an accompanying sympathetic paralysis. Many such cases may have escaped close observation. It is claimed that the lighter iris renders the eye liable to a chronic cyclitis, while others are equally sure that the cyclitis precedes the decoloration; in fact, produces it. In support of either opinion, we have no real proof. From our clinical observation and experimental study the corneal deposits which often accompany the heterochromia should be regarded as a symptom of a disease, and where a sympathetic paralysis is associated, it may properly be reckoned as the cause, producing the passive hyperemia of the iris and ciliary body which Sym and Scalinci describe. Precipitates have been found on the posterior surface of the rabbit's cornea, and I have observed a turbid aqueous after an excision of the sympathetic ganglion, and if we could at will observe our clinical cases, I believe we would find similar changes at some time. To my mind there is much to be said in favor of Scalinci's theoretic consideration of the cause for these precipitates and the formation of cataracts. He maintains that there are abnormal substances which percolate through an abnormally pigmented iris and ciliary body due to vasomotor paralysis. The normal aqueous does not contain albumin, but when the anterior chamber is emptied, that which is then formed contains it, and fibrin may be demonstrated. This rapidly formed fluid is regarded as a transudate from the superficially placed vessels in the ciliary body, rather than a secretion of the body itself, and it is produced by a lowering of the intraocular tension. This change may account for the cloudy aqueous and precipitates noted in rabbits, and for the precipitates seen in man.

The following ocular changes have been produced experi-

mentally: first, a decoloration of the iris (Bistis, Metznen, etc.); secondly, a cloudiness of the aqueous (author); thirdly, the formation of corneal deposits (Bistis); and lastly, uncertain lens opacities. The experiments have not been continued long enough to note more decided lens changes; in fact, it might take years for them to develop—far beyond the natural life of the rabbit.

This, however, does not explain the other type in which there is no evidence of a sympathetic alteration, and yet clinically we have the same picture, namely, heterochromia, precipitates, and finally cataract.

It is, as Dor expressed, an unknown disease. It may and usually does begin in youth and have the train of symptoms described by Butler, but hardly as severe and usually without the serious complications. From the investigations of Fuchs and others it is not a true inflammatory type of disease, such as iritis or cyclitis, and its precipitates may be produced from the same type of passive congestion which takes place in sympathetic disturbances. The origin appears to be in the vessels themselves, a type of sclerosis with obliteration. Whether or not this vasomotor disturbance is of trophic origin, due to a sympathetic alteration without external evidence, is only a suggestion which other investigators of more original thought might consider.

In regard to the formation of cataract, it also appears that its origin is especially due to a lack of nutrition or to altered changes in the aqueous.

Finally our investigations have led us to make the following conclusion:

(1) That the difference in the color of the irides should be called heterochromia iridis.

(2) That in a small number of cases it is inherited.

(3) That in all other cases it is a symptom of some abnormal ocular state.

(4) That in a large percentage of the cases a paralysis of

the cervical sympathetic is the responsible cause for this heterochromia through its trophic disturbances. This paralysis may be inherited.

(5) That besides a congenital heterochromia there is an eye disease of unknown origin, which begins in heterochromia and ends in cataract.

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A STUDY OF THE CONJUNCTIVAL CIRCULATION, TO DETERMINE EARLY SIGNS OF ARTERIO-SCLEROSIS.*

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In the fall of 1911 my attention was called to the possibility of studying the circulation of the conjunctiva by means of a Zeiss corneal microscope. I then began a systematic examination of cases as they presented themselves, paralleling the conjunctival observations with those of the retinal vessels, the blood-pressure, and often a thorough physical examination. To do this work satisfactorily I soon realized that a better method of illumination must be found than the one supplied with the Zeiss corneal microscope. Luedde† describes a light for this purpose, but I did not find it satisfactory. With the help of Mr. C. H. Collman and Mr. DeZeng I succeeded in constructing a light suitable to the requirements of these observations. A small hair-pin filament tungsten lamp was mounted in a handle, to which was attached a system of adjustable lenses so that the light could be concentrated on a small area. The light was not attached to the instrument, but held in the hand; this gave more freedom of movement and consumed much less time. A rigid head-rest was also necessary so that the eye could be held in a fixed position. The A–3 objective and No. 2 eye-piece gave the best image, although the circulation can be seen with a lower powered objective.

After repeated observations and learning what to look for,

* Candidate's thesis for membership accepted by the Committee on Theses.
† Archives of Ophthalmology, 1911, p. 373.

I have come to the conclusion that the normal picture of the vessels and circulation is as follows: Through a transparent wall that is regular in its lumen, the blood-corpuscles are clearly seen. Their flow goes forward promptly and without clogging or stagnation. The rate of flow is not the same in all vessels, neither are all of the very fine capillaries full at the same time. Occasionally one is seen to flash out into view and then disappear, *i. e.*, the corpuscles fill the fine vessels, move on, and an interval passes before the corpuscles fill the vessel enough to be seen. The vessels themselves are not overly tortuous.

The picture of beginning capillary fibrosis is one where the corpuscles clog the lumen of the vessel, forming, as it were, a mass like a log-jam, which holds back the blood-stream. In front of this jam the vessel is empty and for a moment bloodless; suddenly this jam gives way and the circulation goes on. In other fine vessels a bead formation of corpuscles occurs in the lumen which forms an interrupted chain, often remaining nearly stationary for several seconds. This represents stagnation. I have timed some of these, and have counted fourteen seconds by the stop watch before the flow commenced again.

A like phenomenon is seen in an embolus of the central artery of the retina, where a known obstruction exists but is not quite complete; the blood column is seen in some branches to be retarded to the point of stopping for an instant; when an impulse of sufficient strength (the vis à tergo) propels the column of blood onward with a jerky lethargic motion. This same observation is paralleled in observing the conjunctival circulation in capillary sclerosis of the advanced type.

In more advanced cases the capillaries remain nearly empty, and occasionally one may be seen that has become entirely occluded. The lumen of the vessel becomes deformed, resulting in alternate strictures and dilatations not

unlike a string of sausages; these are minute aneurysms. The vessels are tortuous and prominent. The vessels of one eye may show obstructions and dilatations, while the vessels of the other eye may show a nearly normal circulation. This proves, what has often been stated, that vascular change does not progress equally in all vessels of the body, but naturally, when found in one part, all parts are apt to be progressing in the same pathologic direction. When this condition of obstructed flow and stagnation is well marked, the larger vessels show a thinning of the blood column, as though the obstructed tributaries cut down the volume of the current. The color of the blood column also becomes noticeably changed in advanced cases; the lessened volume in the capillary lumen looks lighter red. Where the obstruction is pronounced, the vessel thicker, less transparent, and the volume thinner, the color appears as a rusty brown. In these advanced cases the sclera appears overly white. These changes in the appearance of the capillaries are to be looked for whenever the volume of blood is lessened by constrictions and obliterations.

The condition of the conjunctiva may be taken as an index of the condition found in the capillaries of the skin, and accounts for the pallor of many advanced arteriosclerotics—the characteristic patriarchal pallor of advanced years.

Do the vessels once observed to have undergone these aneurysmic changes ever return to a normal or to a nearly normal lumen? In younger subjects I have seen this. Where faulty methods of living are corrected, the dilatations do seemingly disappear. The following case is an illustration:

Dr. G. A., aged thirty-three, though a practicing physician, spent most of his time in the prescription department of his drug-store, consequently getting very little exercise. He had gained greatly in weight, and was a voracious eater. In many members of his family there was a history of apoplexy, nephritis, and vascular degenerative disease. He had

increasing headaches, aggravated by using his eyes. Examination revealed a compound hyperopic astigmatism. His deep eye showed nothing abnormal other than the fact that the retinal capillaries were quite tortuous. The conjunctival vessels were also tortuous, showing many irregularities and dilatations. The urinalysis was normal. The systolic blood-pressure was 155, with a diastolic of 103. A complete revolution in his method of life, more exercise, less food, cabinet baths, etc., reduced his flesh and brought his blood-pressure down to 125. While the conjunctival vessels remained tortuous, the dilatations that had formerly been so numerous were few and hard to find.

Dr. W. H. Luedde, of St. Louis, in 1912* published a paper on the "Circulatory Phenomena in the Eye." He described the conjunctival circulation as observed with the Zeiss microscope, describing the dilatation of vessels and the usefulness of this observation as a means of detecting early signs of arteriosclerosis.

In May, 1913, a paper was published by the same author† on "A Microscopical Study of the Conjunctival Vessels." In this he goes quite extensively into the description, pathology, and deductions from many observations. He speaks of the regurgitation and reverse flow sometimes seen where the corpuscles go forward; the flow slackens for an instant, stands still, and flows again forward in the original direction.

I have observed this phenomenon, and, like Luedde, I am at a loss to give any satisfactory explanation. Is this possibly the result of friction of the corpuscles contained in a vessel, so placed at right angles to another vessel, that the action would be similar to that of air blown across the end of a tube in an atomizer? Its presence does not seem to indicate any pathologic change.

Pathology.—Koster, in his studies of serial sections made of injected material, found that the degenerative lesions are

* Amer. Jour. of Ophth., August, 1912.

† Amer. Jour. of Ophth., May, 1913.

always first seen around the vasa vasorum, as an infiltration surrounding these vessels, consequently lessening the nutrition to the vessel-walls, promoting secondarily thickening, and interrupting the flow of blood. Other vessels would receive too much blood, consequently causing dilatations and irregularities.

I have made serial sections of the conjunctival vessels. The material was taken from the subject after a thorough study had been made of the lumen of the vessels, the condition of the corpuscles, and their flow in the vessels as observed with the Zeiss microscope. These same vessels, as far as possible, were taken and prepared for the microscope; the section was stained by the Van Gieson method, and showed an increase in connective tissue. Sections of the conjunctival vessels are hard to make for comparative study because of the impossibility of always getting the section straight across, but enough was found to convince me that some changes could be seen. At least there is a difference between the normal vessels before making the sections and the findings after preparation of the sections.

Dr. Luedde found the same difficulty in making serial sections. In his last article he speaks of the changes noticeable in the conjunctival vessels of syphilitics, giving the results of many observations. He speaks almost entirely of the lumen of the vessels, whether dilatations or obstructions are present. In fact, it was the examination of these young luetic subjects that drew my attention to the fact that the action of the corpuscles was the thing to examine for the earliest manifestations of vascular change. These changes are found before dilatations appear.

Mrs. H., aged twenty-five years, had contracted lues about four years prior to her visit. She complained of headache and difficulty in seeing. V. R. E. = 20/50; L. E. = 20/40. The sclerotic was noticeably white. The right pupil was normal and reacted to light and accommodation; left reaction

sluggish; the nerve was white and the vessels small. She had been given salvarsan intravenously, mercurial inunctions, and iodids. The conjunctival examination with the Zeiss microscope showed very fine capillaries with obstructed flow of corpuscles, many beaded vessels with long, empty spaces. There were no dilatations, and the larger vessels showed less volume with rapid flow and rusty red color. The hemoglobin was 85 per cent., showing that the pallor was not from deficient hemoglobin.

Mrs. M., aged twenty-nine years, with interstitial keratitis; luetic infection primary; Wassermann ++. When first seen, the conjunctival vessels were studied, but not reported until the inflammation subsided, the cornea clear, and the Wassermann negative. Here the small vessels showed a marked stagnant flow, with long stretches of beaded vessels; while the large vessels exhibited a rapid flow but no dilatations of the walls.

Miss J., aged eighteen years, complained of poor vision: R. E.=4/200; L. E.=8/200. The cornea showed the scars and obliterated vessels of a past interstitial keratitis. She had the degenerative marks of congenital lues; Wassermann +. The fundus showed spots of atrophy of the choroid from past inflammation. The sclera was noticeably white. The conjunctival circulation showed obstruction and stagnation in the fine vessels with a few minute dilatations.

These cases are only illustrative ones of the early changes in the vessels of luetics. Whether salvarsan and mercury, acting as toxic agents, play any part in causing vascular hyperplasia, it is hard to say, but the examination of the conjunctival vessels of luetics gives valuable data.

A few histories of senile vascular changes noticed in the conjunctival vessels may be interesting and instructive:

Mrs. M., aged forty years, came for relief from headache, a general uncomfortable head feeling, and weariness on exertion. A compound hyperopic astigmatism was found; the eye-grounds and retinal vessels were normal. The conjunctival vessels showed numerous fine dilatations and stagnant beaded flow in the fine vessels. The blood-pressure was

systolic 112, diastolic 80; hemoglobin 70 per cent. The findings were explained to her family physician, who changed her mode of living. Later she reported a great improvement.

Rev. D., aged forty-one years, complained of occipital pain. There was a family history of many members dying of nephritis. He was found to have a moderate compound hyperopic astigmatism. The fundus of each eye was normal. The systolic blood-pressure was 105, diastolic 70. The conjunctival vessels and circulation showed many aneurysmic dilatations, also many irregularities. The corpuscles showed a rapid flow in the large vessels, stagnation of flow and blocking in the fine vessels. The attention of his family physician was called to his condition. A faint trace of albumin was found on several occasions.

Mrs. A., aged sixty-four years, gave a history of years of hard work in an office and the responsibilities for the care and support of several children. Twelve years ago she had a paralysis of the left side of face. The systolic blood-pressure was 212, the diastolic 90. The retinal arteries showed the silver wire appearance, and the veins were indented by the overlying arteries. The conjunctival circulation showed many stagnant, beaded irregularities in the lumen and the small terminals many obliterations. The capillaries were beaded, with wide spaces between the corpuscles; further on a corpuscle or two that were absolutely stationary, and beyond this a faint line showing the presence of the obliterated vessel.

The following case is of interest, as it gives a picture of polycythemia:

J. A. S., aged fifty-five years, consulted me for presbyopia and an eye examination at the request of his family physician, who reported red blood-cells, 6,700,000; hemoglobin, 120. The retinal vessels were normal. The conjunctival circulation showed many aneurysmic dilatations; vessels quite tortuous and noticeably crowded by corpuscles; a number of the capillaries showing stagnation and blocking, and a very rapid flow in the larger ones.

Sufficient organic changes to be called pathologic have been

found in the conjunctival circulation of young pregnant women who were suffering from the toxemia of pregnancy.

These case reports are a few types I have chosen from many, wherein degenerative changes in the conjunctival vessels were observed, when nothing significant was to be seen in the retinal vessels.

With the pocket loupe or the Coddington lens of over ten diameters of magnification, and using a common battery flash-lamp as a means of illumination, a good idea of the general vascular system can be determined: whether the vessels are overly tortuous or have aneurysmic dilatations. In subjects who have marked slowing and thinning of the blood column in these vessels even the circulation can be observed.

Whenever the internist suspects vascular degeneration, he should examine the conjunctival vessels as a routine measure. He will find changes here long before there is any significant change in the appearance of the retinal vessels.

I have demonstrated this fact to one of my medical friends who does diagnostic work, and he is convinced, after trying this method for over a year, that much more and earlier information is to be had than by the use of the ophthalmoscope.

The collective observations of many men should be made with the larger instrument, and record made of the action of the corpuscles in the blood-vessels under the action of various drugs locally and internally administered; whether certain drugs, like salvarsan, mercury, etc., have any action on the vessels. The result of fatigue and its toxins should also be studied.

Summary.—With the aid of a Zeiss corneal microscope and a light designed to focus a small spotlight on the scleral conjunctiva, the conjunctival vessels outlined by their contained corpuscles show a very early vascular change not demonstrated in any other part of the body. These changes are manifested by a clogging of the corpuscles in the lumen of the vessel; in front of this the vessel is empty for a short

space. In other fine vessels a beaded formation occurs in the lumen which forms an interrupted chain, representing stagnation of the flow. In advanced cases the vessel may be nearly empty and at its terminal extremity show complete occlusion.

The condition found in the vessels of the scleral conjunctiva is a good index of the general vascular system.

THE VITREOUS* AND ITS REPLACEMENT AFTER LOSS.†

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Historic.—In the pre-ophthalmoscopic days, much was conjectured concerning processes in the posterior part of the eye. Consequently attention was given to those things and parts in the living which could be directly seen and observed. Hence ophthalmologic text-books possessed lengthy accounts of the ophthalmias with little relating to what now are considered very important subjects. Truly, then, did Helmholtz work a revolution with the discovery of the ophthalmoscope. Up to his time little space was given, as a rule, to a consideration of the vitreous other than by anatomists. Demours and Zinn, early in the eighteenth century, had studied this structure and furnished us with data of inestimable value. Following shortly the studies of Bowman, Arnold, Brücke, and Hannover, came the epoch-making discovery of the ophthalmoscope. This gave a tremendous impetus both to clinical and histologic investigations. The early ideas regarding the vitreous were rather obscure and even at times fanciful. It was at one time looked upon as a

* The adjective "vitreous" is used in the sense of a noun, the word "body" being understood.

† Candidate's thesis for membership accepted by the Committee on Theses.

transudate and not as a structure. In recent years it has advanced from a tissue to the dignity of an organ (Tschern-ing, Stilling).

Probably the earliest description of the vitreous handed down to us unimpaired is that of Galen, who lived in the second century after Christ. Galen's description remained the best for many centuries. His conception of its function, however, widely missed the mark, as he supposed an intimate association existed between the retina and vitreous. Of necessity all early observations were macroscopic, but with the advent of the microscope a new era began. It was not until the middle of the nineteenth century that careful studies of the minute anatomy of the vitreous were made. Early writers referred to three humors of the eye—the aqueous, crystalline, and vitreous. As late as 1805 Ware repeated this statement. At all times the vitreous had been regarded by ophthalmologists, with a few notable exceptions, as having only a passive mechanical function. With the investigations of Kölliker, Bowman, and Virchow, a more accurate understanding of the structure of the vitreous began. The former writer looked upon the vitreous as a gelatinous connective tissue, whereas the latter classified it among the mucous tissues. Leber designated it as a sort of hydropic or watery connective tissue.

Embryology.—As Greeff well remarks, about no part of the eye does there exist such a difference of opinion as there is concerning the genesis and histology of the vitreous. The origin of the vitreous needed explanation, and its derivation from the mesoblast furnished a most convenient logical and enduring theory. This stood the test of time, and was not viewed with suspicion until about 1898, and since then, though vigorously attacked, it has not been completely dislodged. The early conceptions regarding the origin of the vitreous had to do almost wholly with the mesoblast. This theory of Schöler gained universal attention and held un-

disputed sway for many years. It was then seriously questioned and many facts adduced to weaken its position. Advocates of the epiblastic theory arose and these became in time subdivided. One set of observers held that the vitreous was of epiblastic origin, springing from the retina, especially from the anterior portion. Others subscribed to the epiblastic theory, but looked upon the lens, an epiblastic structure, as the source of the basal substance of the vitreous. Still others in recent years have ascribed its origin to both the lens and the retina. At the present time a number of writers "straddle" the proposition and think that both epiblast and mesoblast are concerned in the development of this structure (Van Peé, Kölliker).

The vitreous space in the early embryo is inconsiderable, the greater part of the secondary optic vesicle being occupied by the primitive lens. The primitive vitreous substance is of short duration. In 1898 Tornatola directed attention to the ectodermal origin of this structure, and corroborating this contention in the main were Rabl, Addario, Fischel, Kölliker, v. Lenhossek, Wolfman, Salzmann, and others. Van Peé believed in a mixed genesis, the mesodermal factor predominating, whereas Kölliker, while agreeing to a combined genesis, looked upon the ectoderm as the principal factor, the mesoderm playing a much less important part. The ectodermal theory, while generally accepted of late, was opposed by a few authors who revived the mesodermal theory or refused to accept the new. Nussbaum adhered to the mesodermal origin and positively stated that with the exception of the lens, retina, and pigment layer of the iris, all the tissues of the eye are developed from the mesoderm. It appears that this latter author dismissed the subject with scant consideration of the contentions of differing writers. A predominant part is attributed to the neurogliar cells in the formation of the vitreous both solid and fluid. The vitreous fluid and the aqueous humor are of identical origin,

at least during the development of the eye (Magitot and Mawas). In an earlier communication these latter authors divide the vitreous development into three stages: (1) The primordial vitreous; (2) the transitory vitreous; (3) the permanent vitreous. The first is ectodermal and is for a time obscured by the intrusion of the mesodermic hyaloid vascular system. This latter does not participate in the formation or constitution of the vitreous. It occurs in the fourth week, and its rôle is solely vasoformative. At the eighth or ninth week the transitory vitreous appears. Its neurogliar cells secrete the vitreous fluid and its life is short, becoming displaced and compressed from the third month on, finally constituting the central vitreous. At birth only traces remain. At about the tenth week the primordial vitreous takes on renewed activity throughout the whole retinal extent. This lasts up to the fourth month, and from then on the formation of the vitreous is a function of the ciliary retina. The abridged views of Wolfman are the following:

(1) The lens is not concerned in the development of the vitreous. (2) The vitreous body in mammals is purely of ectodermal origin, arising from the retina. (3) The mesoderm, especially of the vessel system, has only a nutritive function in the development of the vitreous. It is obvious that the question, especially in its finer details, is still unsettled; that the theoretic stage has not been passed, and that concrete and indisputable facts are still to be brought forth before the matter is definitely decided. With few exceptions, however, the consensus of opinion is that the mesoderm forms only the vitreous vessels and that the framework of the vitreous is of ectodermal origin (Salzmann).

Anatomy.—The anatomy and structure of the vitreous have for a long time been in dispute and the subject of widely diverging views. Stilling cogently remarks that the anatomic examination of the eye must form the basis of all knowledge

of the physiologic processes, for a correct presentation of the action of any mechanism must rest upon a proper appreciation of its structure. The vitreous occupies in the human about three-fourths of the capacity of the eyeball. Its consistence is jelly-like, and it is by many looked upon as inert or at most having but little other than a passive mechanical function. All, however, have not subscribed to this view, Stilling, for instance, crediting it with an important active part in the function of accommodation. Tscherning, Stilling, and others refer to the vitreous not as a structure, but as an organ having a special function. Magitot and Mawas define the vitreous as neuroglia adapted to optical function. With age the fibrillar framework of the vitreous probably becomes less dense.

As a rule, the earlier ideas pertaining to the vitreous have been supplanted or much modified. Formerly the vitreous was thought to be divided by septa, situated either radially or concentrically. Thus arose the "orange and onion" theories. The former was disproved by the fact that after puncture of the cornea all the fluid will drain away if the eyeball be suspended, which would not be the case were the vitreous divided into sectors like an orange. The histologic examinations also refute this contention. Likewise the lamellar or onion theory of structure was contested, it being impossible to demonstrate microscopically any such lamellæ. True, condensations of the vitreous centrally and peripherally might simulate layers, yet that a true lamellation or onion-like formation obtains in the human vitreous is pretty well disproved. The combined radial and concentric structure is by others attributed to the vitreous, the periphery being concentric, while toward the center a radial arrangement is assumed. This is highly hypothetic, and may well be doubted.

The existence of structural elements consisting of fibers and cells was shown by Kölliker and verified by Virchow.

This constitutes but a very small part of the vitreous, being variously estimated from .03 to .07 part by weight. The presence of isolated living cells, so-called subhyaloid cells, are noted. They are wandering cells and possess ameboid movements. It is uncommon for these cells to be round. As a considerable interval exists between them, they do not form a continuous coat. Salzmann describes the fibrillary structure as growing out from the base of the vitreous, a point adjacent to the ora serrata; a part flows backward in a fine, wavy striation to form the posterior border layer (rind). There is a somewhat smaller anterior border layer. This characteristic formation is likened to the flowing hair of a woman. It has also been compared to the flowing tail of a horse (Pferdeschwanz). The remainder radiates fan-like into the interior, forming the loose structured body of the vitreous. The thicker rind or border layer noted by Salzmann is absent at but two points—the optic nerve entrance (area martegiani) and anterior at a point between the base of the vitreous and the margin of the anterior border layer. It has not been established whether or not these areas are efferents for the fluid of the vitreous, as has been claimed. The later writers have looked upon the vitreous as a fibrillary network filled with a watery fluid—a fibrillar sponge, as it were.

One is struck by the emphasis of dissenting views regarding the existence of a hyaloid membrane. Two such writers as Greeff and Salzmann voice diametrically opposite opinions. The former asserts the presence of a hyaloid membrane as distinct from the membrana limitans interna, and that a lymph-space intervenes.* The latter denies the existence of more than one membrane (membrana limitans interna). The existence of a definite capsule of the vitreous, the hya-

* Burdon-Cooper found, by entoptic methods, a fine mosaic, the elements of which possessed movements. This mosaic was thought to be occasioned by a thin layer of fluid existing between the retina and the hyaloid membrane, which argues for a lymph-space present between these structures.

loid membrane, is generally accepted, though many writers (Iwanoff, Salzmann, Merkel, Magitot and Mawas, and Kölliker) have denied its existence. Some of its advocates have claimed its presence at all points except the anterior surface, the patellar fossa. A distinct membrane here does not exist, but is represented by a condensation of the fibrillar structure, a virtual membrane resulting. Rather firm attachment of the vitreous occurs at but two places, the optic disc and pars ciliaris retinæ. This latter attachment is quite firm. The membrana limitans interna may be considered a part of the retina or vitreous, as one chooses; but Salzmann emphatically states that no second membrane exists between the bases of the Müller's fibers and the border layer of the vitreous. The anterior rind or border layer is frequently described and mistaken for a distinct hyaloid membrane. The hyaloid membrane, about which there has been so much dispute, is probably but a condensation of vitreous substance. The same is true of Stilling's canal, which is present only in mammalians; in birds and reptiles it is represented by the pecten and in fish by the processus falciformis. The debate about the existence of Stilling's or Cloquet's canal is no less spirited than that concerning the embryologic origin of the vitreous or the presence of a hyaloid membrane. Eminent writers vouch for its existence and produce experiments and proofs and even stereopticon pictures (Bribach) defending the contention. Equally eminent observers deny its existence and view the proofs as faulty and the conclusions as erroneous. A wall is claimed for the canal by some and by others denied. At the most, the so-called wall may be looked upon as merely a condensation of the fibrillary structure. Best, among others, denies the existence of a distinct central canal, though a physical canal is accepted. The claim that the canal of Stilling, the pecten of the bird and the processus falciformis of the fish are homologous is denied by Franz.

Composition.—The composition of the vitreous differs from the aqueous chiefly in an apparent slight excess of

albumin in the former. This was an early contention of Deutschmann. Later observers have denied any difference in the albumin content. The slight difference between the composition of vitreous and aqueous may be explained on the theory of slower metabolism and fluid exchange. The albumin content and the salts show very little variance. The specific gravity of each is approximately 1008, and the osmotic coefficients differ but slightly, being for aqueous 0.96 by the blood-corpuscle method, and 0.925 by the freezing-point method; the figures for the vitreous being in the respective methods 0.971 and 0.959. The albumin content is very small, and is composed of serum-albumin and serum-globulin. The quantity of albumin has given rise to some discussion, Deutschmann contending that the vitreous is richer in albumin than the aqueous. This might be due to the methods employed, some investigators figuring in mucin with the albumin estimate. Dogiel states that a trace of albumin is always present in the vitreous and that it is hardly to be concluded that this comes from the albumin either of the retina or lens. Both aqueous and vitreous resemble cerebrospinal fluid very much in their composition. If vitreous is filtered, about 99 per cent. passes through and 1 per cent. remains on the filter, this latter being made up of the cells and tissue substance (framework) of the vitreous. The reaction of the vitreous is alkaline. The inorganic content amounts to about 1 per cent. and is composed of the following salts—the percentages both of aqueous and vitreous being presented for comparison (Parsons, vol. iii):

	Aqueous	Vitreous
Sodium chlorid	78.11	74.43
Sodium carbonate	8.72	12.67
Potassium chlorid	2.92	5.57
Potassium sulphate	5.99	3.74
Sodium phosphate	1.99	1.82
Calcium phosphate	.62	.44
Magnesium phosphate	.40	.22
Total	98.75	98.89

None of the refracting media of the eye exceeds the vitreous in transparency. It has an index of refraction of 1.334, corresponding closely thereby to aqueous and water. The vitreous maintains its transparency a long time after death (Salzmann). After it is heated for five hours at a temperature of 108°, the fibrillar structure is dissolved. The vitreous possesses considerable elasticity, as shown by experiments with metallic quicksilver. While this experimentation was not repeated by us, the general impression was gained that elasticity is a conspicuous quality of the vitreous. It has been thought that the density of the vitreous differs in its different parts, being greatest at its lowest and least in its upper portions (Burdon-Cooper).

Source of Vitreous Fluid.—The contemplation of the source of the aqueous and vitreous fluid is a fertile field for speculation. While at the outset it was not intended to take sides or commit oneself to a given theory, still convictions could not be evaded or ignored. Upon a clear basis of understanding regarding the arguments pro and contra, relative to this fluid, whether it is a stagnant pool or an ever-changing and ever-renewed stream, rest many of the theories and explanations regarding other physiologic and pathologic ocular phenomena. A strong point in favor of the ciliary origin of the vitreous fluid is the dissipation and shrinking of that structure upon the experimental removal of the ciliary processes (Deutschmann). These experiments were repeated by Nicati. The removal of the ciliary body alone is impossible in the rabbit, as the processes grow on to the back of the iris, but it is possible in the dog. The ablation of the iris alone has no effect on the vitreous. Nicati designated the two layers of epithelium covering the ciliary processes as the aqueous humor or uveal glands. The existence of the so-called Treacher Collins ciliary glands has never been established. The glands of Collins were described as protrusions from the pigment epithelium toward the ciliary muscle.

These processes, composed of cells, were held to show a tubular arrangement on cross-section. Alt was unable to confirm this tubular structure. Rutteman also failed to demonstrate a lumen. Finnoff found a lumen in but one specimen of the many he examined and this was thought to be an artefact. He states that a gland-like structure composed of pigment epithelium situated deep in the substance of the ciliary body is highly improbable, and that the aqueous and nutrient fluid of the vitreous are probably secreted by both layers of the epithelium, which cover the ciliary body from the ora serrata to the root of the iris, the pigment epithelium playing, most likely, the more active part, because of its location. Mawas endeavored to prove that the cells of the epithelium of the ciliary body are glandular in character. This faculty was attributed to both the unpigmented and the pigmented layer. As proof of this contention, there was offered the evidence of the occurrence of secretory granules and of variations in the chromatin content of the nucleus of the cell. The formation of an unstained fluid from a stained blood in a tissue loaded with staining matter, the epithelium alone remaining free from color, can only be attributed to the action of the epithelium (Nicati). It is held by Magitot that the aqueous is a stagnant pool, and what is absorbed is replaced by a process of dialysis by the retinal neurogliar cells, *i. e.*, the cells of Müller's type, which are cells with a vitreous prolongation, the cells of the ora serrata and the clear ciliary cells, whose exoplastic prolongations form the zonule. The one fact which has proved an obstacle to the filtration theory of the production of aqueous is the higher sodium chlorid content of aqueous as compared with blood plasma: this apparent discrepancy, however, is not difficult of explanation.

Circulation.—It has been stated (Leber) that the source of the aqueous and vitreous fluid, at least in mammals, is from the ciliary processes. However, the foundation for this conclusion was based principally upon the fact that the vitreous

shrinks upon removal of the ciliary processes and upon injection experiments in animals. Because of the element of diffusion, reliable conclusions cannot be drawn from positive findings in which substances, as potassium iodid, are experimentally injected into an animal's circulation. In this connection only negative findings are of value.

Various theories advanced regarding the production of aqueous have been filtration, osmosis, dialysis, and secretion, the last a vital process. O. Weiss is strongly opposed to the view that a continuous lymph-stream exists in the posterior and anterior chamber and Schlemm's canal. Franz, Magitot, and Mawas are likewise inclined. All experiments tending to prove the existence of a continuous current were shown to have been made on eyes with a zero tension.

The permeability of the zonule of Zinn to aqueous fluid has been adequately demonstrated (Gifford, Pflüger). India ink and other substances injected into the vitreous have found their way forward. Air injected into the vitreous for the cure of detached retina (Ohm) soon appeared as bubbles in the anterior chamber, but was quickly absorbed. Hess has conclusively shown that no difference of hydrostatic potential is to be detected between various parts of the eye. Manometers connected with the vitreous and anterior chambers stand at the same level.

For a fluid to be formed against a pressure approximating or exceeding the blood-pressure implies a vital force or function, namely, secretion. By some the tubular outgrowths of epithelium in the pars plana have been considered as specific secretory glands engaged in the secretion of the aqueous (Treacher Collins). Intravascular tension must exceed extravascular pressure if blood circulation is to continue. In this connection it has been pointed out (Weiss), with regard to the venæ vorticosæ as they pass through the sclera, that the venous pressure here must exceed the intraocular pressure. The pressure in Schlemm's canal then is still higher

than in the vorticose veins, and is therefore higher than intraocular tension; consequently fluid cannot flow from the anterior chamber into Schlemm's canal. Such is Weiss's contention, which assumes the characteristics of a mathematical equation. If a is greater than x and b is greater than a, then b of necessity is greater than x. Whether the foregoing argument is fallacious or based upon false premises, one cannot state with certainty at present. Still, absolute faith in the older and long-accepted theories is rudely shaken. The fluid is formed, however, not against a high pressure under normal conditions, but, if Weiss is correct, it is by the older theories excreted against a higher pressure. This is manifestly impossible, and one is led to conclude that either Weiss's reasoning is wrong, or the older theories are wrong. Wessely's views regarding the intraocular exchange of fluid differs but little from those of Weiss. The theory that the iris is truly concerned in the formation of aqueous (Ehrlich, Hamburger) and that the ciliary body is a secondary consideration has been completely disproved, chiefly by the work of Leber. The assumption that the venous radicles of the iris are concerned in absorption of the aqueous appears untenable. For fluid to pass from the anterior chamber into the iris veins, the intraocular tension would of necessity have to be greater than the intravenous pressure (Uribe y Troncoso). Were this so, the veins would collapse.

The metabolism of the vitreous is very slow, yet as some is excreted, some is renewed. The route by which it leaves the eye has been the subject of discussion. Schlemm thought it escaped by way of the optic disc along the optic nerve. Very little, probably not over 2 per cent., is removed in this way. The remainder passes out of the eye at the angle of the anterior chamber. Postmortem secretion of aqueous does not occur, and it is most probable, therefore, that the passage of fluid into the vitreous cannot happen after death in an eye which has previously had its vitreous chamber tapped.

Function of the Vitreous.—An important function of the vitreous is the support of the retina, another the refraction of light. Tscherning has made the interesting and important point that the vitreous is an organ, and not a tissue or a bag filled with water. Stilling believes the vitreous to take an important part in the phenomenon of accommodation.

Immunity: Antibodies.—This chapter opens a wide field for further endeavor. Some important work has been done by Bürgers and others. One is tempted to go into a discussion of this interesting subject, but it is hardly relevant to the matter in hand, as it borders on the pathologic. A few words may be said in passing. Bacteria find in other tissues much better pabulum, but are deterred and restrained by the presence of sufficient antibodies. The vitreous is relatively barren, antibodies likewise are few, and because of the slow fluid exchange they are extremely tardy in reaching an assailed vitreous focus. A lack of vascular supply necessitates a roundabout passage for antibodies. On the other hand, as a result of irritation, the aqueous becomes rich in antibodies coincident with an increased albumin content. Ordinary irritation, such as would produce an increase in aqueous albumin content, and, *pari passu*, an increase of antibodies, does not react in like manner with the vitreous, but the response here is slow and imperfect (Bürgers). As a result of experiments in guinea-pigs, Salus concluded that all antibodies without exception pass over into the aqueous of the healthy eye from the blood. On the other hand, Possek was of the opinion that the bacteriolytic substances contained in the blood-serum are absent in the vitreous altogether or exist only in the most minute quantities as long as the secretory apparatus of the eye is intact.* Kuffler's experiments to

* To tap the vitreous, for instance by trephining, as a therapeutic measure when that structure is infected, while possessing some theoretic advantage, might prove a double-edged sword. Both antibodies and albumin content would be increased, and should the former not predominate, fresh pabulum would only be furnished to the infecting agent.

determine the presence of immune bodies in the vitreous showed that these bodies did not pass from the general circulation of an actively immunized animal into the vitreous in demonstrable quantity.

Vitreous Loss.—The amount of vitreous which may be lost without resulting in retinal detachment or hemorrhage varies between rather well-defined and narrow limits. The rapid removal of more than 0.5 c.c. of the vitreous fluid in the rabbit invites hemorrhage, and in larger amounts detachment of the retina. The eye after aspiration apparently regains its normal tension in two or three days, if the amount of fluid withdrawn be not excessive. The tapping may be repeated at intervals without seeming damage. Schreiber removed from one rabbit's eye in the course of two months a total of 3.9 c.c. of vitreous fluid in five sittings. We did not investigate the effects of repeated tappings. In some respects we are in agreement with Schreiber's further findings. He detected no notable change in the vitreous, ciliary body, iris, or choroid, and we are led to the conclusion that this is correct except with regard to the changes in the ciliary body. In this body pronounced changes were found by us comparable to those following paracentesis of the anterior chamber. The changes, though marked, did not reveal an unusual number of Greeff's cysts. A matter of much importance is the time at which the specimen is taken for microscopic examination following the experiment. With a restoration of the volume of the vitreous, there occur of necessity a diminution and finally a termination of the activity of those factors and forces making restoration possible. As the eye fills in two or three hours after tapping, the changes in the ciliary processes, if these be the source of vitreous fluid supply, begin probably to recede. Hence eyes examined after several days show little or no change. In our investigations, eyes examined a week after loss of vitreous showed virtually no changes in the ciliary processes. Experiments

on rabbits demonstrate that if more than 0.5 c.c. of vitreous fluid is withdrawn, detachment of the retina is prone to occur (Birch-Hirschfeld, Löwenstein, and Samuels). However, as Birch-Hirschfeld states, the susceptibility to detachment is not nearly so great in the human as in the rabbit's eye. Vitreous once lost is not reproduced as vitreous. The fibrils are incapable of reproduction or division and contain no imperfectly developed elements (Greeff, Bartolotta).

Prologue to Discussion of Experiments.—It is neither the office nor the province of this paper to take up the consideration of the diseases of the vitreous. Its anatomy, embryology, and physiology are pertinent to the subject, and are adjuvants to experimental work having for its object a better understanding of this interesting and important structure. The experimental work was done upon rabbits, this animal being selected as the best and most available for our purposes. It was clearly understood that the ocular structures of the rabbit and human differ markedly in many respects. Yet it was considered permissible to draw certain conclusions, deemed legitimate, from the work undertaken.

The corpus vitreum has been the subject of much study and more speculation. Considering the immense amount of investigation of the eye and its component parts, relatively little is definitely settled concerning the vitreous. Its origin is yet in dispute; its anatomy in detail has not been decided beyond criticism; its function and metabolism are not fully understood, while its regeneration or replacement after loss is a matter of some conjecture, not as to whether it is replaced as vitreous proper, but as to how it is replaced. While not so presumptuous as to hope to clear up any or a single phase of one of these problems, it is my desire to present the results of investigations which, though they are in all probability not new, were yet in their inception original with us. In experimentally studying the replacement of vitreous after loss, collateral information was gained and collateral sub-

jects touched upon. The embryology needed to be taken into consideration, and the lymph circulation of the eye had to be studied. The experiments of Deutschmann had shown that after removal of the ciliary processes the vitreous was absorbed, but that this did not occur after removal of the iris. This demonstrates that a consideration of the lymph circulation is of necessity bound up with a study of the vitreous. While it is generally supposed that the fluid content of the vitreous is supplied and renewed solely by ciliary activity, it has not been definitely proved, neither has it been unanimously accepted, for some think the choroid is an added source of aqueous humor.* The "emergency aqueous" formed after tapping the anterior chamber differs markedly in composition from normal aqueous, and is probably, as Magitot states, a modified serum from the vessels of the ciliary processes with some aqueous fluid squeezed from the fibrillary sponge (vitreous body). If the intraocular pressure is 25 mm. Hg, or thereabouts, and the arterial and venous pressure approximately 70 and 40 mm. Hg, respectively, we would expect, *a priori*, a sudden lowering of the intraocular pressure to 0 to result in a marked circulatory derangement in the vessels of the ciliary processes. This would be anticipated whether the tension was lowered by a tapping of the anterior chamber or the vitreous chamber, and that much the same train of phenomena would supervene in both instances. Two points, therefore, claimed our attention: (1) The establishment of the fact that the same changes occur in the ciliary processes after vitreous tapping as after aqueous (anterior chamber) tapping. (2) The change in composition of the vitreous fluid, shown principally by a marked increase of the albuminous content, such as occurs in emergency aqueous. I recognize that our experiments were lacking in some

* Changes were therefore looked for in the choroid and retina in addition to the ciliary changes in our experiments on animals in which the vitreous chamber was tapped.

features but, on the whole, however, are held to be sound and may be briefly stated as follows: (1) That vitreous when lost is not regenerated as such, but is replaced by aqueous. (2) That immediately after loss vitreous is replaced by an albuminous fluid similar to emergency aqueous when the anterior chamber is opened. (3) That examination of the ciliary processes show changes analogous to those which occur in these structures soon after draining the anterior chamber.

That vitreous is not replaced as vitreous proper is proved by microscopic studies (Greeff and others) and by the fact that it is too quickly accomplished. An avascular structure of low metabolism, such as the vitreous is, would not regenerate rapidly, granting for argument's sake that it could regenerate at all. The fluid which replaces vitreous immediately after loss is held to be emergency aqueous (a modified plasma) by reason of its albuminous nature, the rapidity of its formation, and the accompanying changes in the ciliary processes. The changes in the ciliary processes after vitreous tapping as viewed microscopically differ markedly from the normal and resemble in appearance those changes which follow drainage of the anterior chamber. Two of these propositions are not new, especially the first, it having been long ago demonstrated by a number of observers. The second was stated by Greeff to have been touched upon by Wessely, while that writer was conducting some experiments on ocular lymph circulation.* In so far as the writer is concerned, it was an original observation and was made directly and primarily for the purpose of ascertaining if the albumin content of the vitreous was increased after tapping. The third as a matter of course followed the second. To the time of writing no reference to this has been encountered in a study of the literature except an article by

* On looking up the original article by Wessely, that writer states decidedly that in his experiments the albuminous content of the vitreous was not increased after aspiration of the vitreous fluid. Greeff appears to have misquoted Wessely.

Schreiber, a reference by Nicati, and the allusion by Elschnig* to the experiments of Birch-Hirschfeld. This latter investigator performed numerous experiments on animals to produce and study detachment of the retina. In such instances the subretinal fluid forming consecutively to the detachment was found to be rich in albumin.† Another and the most direct reference encountered relative to the increase of albumin in the vitreous fluid after vitreous puncture is by Nicati, who first injected fluorescin, evacuated a "certain" amount of vitreous through an equatorial puncture and noted a coloration of the fluid. It was less intense and slower to appear than after anterior chamber puncture. "In all these cases secretion was fibrinous."‡ Hence it was concluded that the occurrence of fibrinous secretion is a reflex act consecutive to the lowering of tension in the eye.§

The tapping of any chamber, anterior, posterior, or vitreous, for the time being reduces the intraocular tension to 0.

* Elschnig referred to the beautiful experiments of Birch-Hirschfeld wherein that investigator aspirated vitreous fluid and found the reformed fluid to be rich in albumin. No bibliography was given and no reference made to the article of Birch-Hirschfeld in which the experiments were described.

† In this case the fluid is to be readily sought as coming from the choroid vessels, which are in juxtaposition. There exists a potential cavity between the retina proper and its pigmented layer, the remains of the space between the two layers of the secondary optic vesicle, and it is at this place subretinal fluid collects. The very fact that it is confined in such areas is evidence that fluid from the choroid does not seep or filter through the intact retina, thereby reaching the vitreous. A point to be decided is whether the accumulation of fluid produces the detachment or the detachment causes the fluid to be produced. Substantiating the former, however, is the fact that reattachment frequently occurs with rapidity upon aspiration of the subretinal fluid.

‡ Nicati presumably included in his statement the vitreous tappings with those of the anterior chamber about which he had just been writing in the preceding sentence.

§ Elschnig must have had in mind the replacement of lost vitreous by albuminous fluid. In the substitution of vitreous by saline solution as a therapeutic measure, this author noted that the injection of salt solution not only prevents the rapid exudation of albumin, but by reëstablishing tension exerts a favorable influence on the ocular circulation. This substitution would need be rapid in order to anticipate the pouring out of an albuminous fluid by the ciliary processes. It is the sudden reduction in tension which occasions the transudation of this fluid. Therefore the rapid reëstablishment of tension would minimize the "secretion of the emergency aqueous."

This cannot occur without compensatory changes. We would naturally look for those changes where minor fluctuations are physiologic, viz., the arterial blood-vessels, and where those blood-vessels are numerous and lacking firm support, in the ciliary processes. Engorgement and transudation are to be expected when extravascular tension is suddenly reduced from 25 mm. Hg to 0, with an endovascular pressure of 70 or thereabouts intact. That this albuminous fluid might come from the choroid is hardly probable, for various reasons, but chiefly for the fact that the microscopic appearance does not warrant such an assumption. Fluid from the choroid would be compelled to filter through the intact retina, which could hardly occur without producing appreciable changes in that structure, and these were not noted.* After injection of fluorescin and evacuation of the aqueous, the choriocapillaris alone is stained, the retina remaining absolutely colorless; this result is entirely confirmed by the ferrocyanid method (Nicati). Because of the fact that the vitreous is frequently involved in affections of its neighboring structures, the retina and choroid, it has been assumed that its nutrition comes from those tissues. While it may be fair and correct in this instance to reason from pathology to physiology, other sources, as, for instance, the ciliary processes, cannot be readily excluded. It is quite as logical, or more so, to look to the ciliary body for vitreous physiology and pathology. It was noted by Elschnig that pathologically dense vitreous rich in albumin could with benefit be aspirated and replaced by physiologic salt solution. This aspirated vitreous, contrary to expectations, resembled a blood-serum like flocculent fluid without blood coagulate. It is interesting to speculate on the origin of the vitreous opacities following penetrating injuries, whether

* In experimental detachment of the retina, Birch-Hirschfeld noted that the retina always showed one or more tears. This could easily account for albuminous subretinal fluid infiltrating the vitreous.

they represent always the remains and results of hemorrhage, or whether they are sometimes albuminous flocculi from the transuded fluid originating in the ciliary processes.

Discussion.—Various methods were used in obtaining the vitreous, both from the living normal and dead normal and from the living and dead punctured vitreous chamber. Elements of inaccuracy entered, necessitating either a variation of technic or a confirmation and corroboration. Normal aqueous was removed by syringe from the living animal in some cases; in others a pipet introduced through a small incision was used. The same methods were utilized for obtaining both normal vitreous fluid and "emergency fluid" in dead rabbits immediately after killing them. Whatever method was used, a normal and tapped eye were observed together, as a rule, in order that identical conditions obtained in both instances. A number of eyes and vitrea had to be discarded for obvious errors. The prevention of contamination by blood was the most serious problem. The withdrawal of more than 0.5 c.c. of vitreous fluid was prone to cause hemorrhage and detachment of the retina. In quite a few instances the vitrea were thrown away for this reason.* On the whole, the most satisfactory results were in those eyes enucleated immediately after death, all tissues quickly and cleanly dissected off, and the globe passed rapidly through saline solution and dried. The globe was then grasped at the optic nerve entrance and held by forceps, while a disc of the tunics 3 mm. in diameter was shaved off with a sharp Graefe knife. The globe was then placed over the mouth of a test-tube and the vitreous fluid allowed to drip into the tube. Unless the fluid from the tapped eye was as clear as the

* It is conceivable that the sudden reduction of intraocular tension to 0 by aspirating the vitreous fluid might result in small hemorrhages comparable to the appearance of red corpuscles in the aqueous, which sometimes occurs in draining the anterior chamber. In such a case it could not be designated a true contamination, but rather an aggravated result of the experimental procedure.

normal, it was thrown away. Whatever element of error or imperfect technic entered into this procedure obtained in both the normal and tapped vitrea. The point at issue was solely whether or not the vitreous, after tapping of its contents, was replaced by a fluid with an increased albumin content. This the experiments seemed clearly to indicate no matter what technic was used. The vitreous was so quickly secured that the likelihood of postmortem changes were very remote, yet if they did occur the normal and tapped vitrea were equally affected. The fluid appearing among the lens-fibers after death is a postmortem change and is by some looked upon as an infiltration of the aqueous. Conscious of this fact our observations were made as soon as possible after death, to preclude the chance of postmortem changes influencing the vitreous. With the thought in mind that results might differ according to whether the anterior or posterior vitreous was examined, both were tested with identical results. A further noteworthy finding was that examination of the fluid a week after tapping the vitreous chamber failed to show any considerably increased albumin content. Adrenalin should not be used in anesthetizing solutions in experiments such as were carried out, as it lessens the proteid increase in the ocular fluids following aspiration and stops the formation of Greeff's cysts in the ciliary processes. An interesting point noted in connection with the tapping of the vitreous chamber is that, on removal of the eyes after a wait, the vitreous as a whole of the tapped eye is apparently more watery and less formed and jelly-like than in the normal eye. Vitreous fluid obtained from a number of rabbits' eyes was examined in the Department of Physiological Chemistry of the Washington University Medical School. Because of the disorganization of that department on account of the war, as full an examination as desirable could not be obtained. No quantitative tests were made. However, the presence of albumin in considerable amount was demonstrated in the

vitreous fluid of eyes which had been previously aspirated, whereas but a trace was found in the normal vitreous fluid. The albumin was noted as serum-albumin.

While studying the behavior of scleral wounds in rabbits, a number of eyes were operated upon by various methods (scleral trephining, incision, electrocautery, red-hot needle, seton, sclerocorneal trephining, and iridotasis) and were later enucleated at varying intervals. Opportunity was thus afforded for a study of the changes in the ciliary processes. Observations were made at varying periods subsequent to the drainage operation. In another series of animals tapping of the vitreous cavity, by aspiration, incision, or trephining, was done with the direct object in view of studying the ciliary processes and the fibrillar structure of the vitreous. In all, 27 such eyes were studied. Three eyes in which paracentesis of the anterior chamber was done were observed in connection with these. The interval between tapping or aspiration and enucleation varied from fifty minutes to six weeks. A majority of the eyes were fixed in formaldehyd, hardened in alcohol, and stained with hematoxylin-eosin. The remainder were fixed in Zenker's fluid and stained either with Van Gieson's stain or with hematoxylin and eosin. A few were depigmented to better study the changes in the ciliary processes. Without depigmentation it was found that the observations were somewhat impeded in the fully pigmented rabbits, whereas in the white or albinotic animals the study was much more simple and satisfactory. This was particularly true in searching for Greeff's cysts. The finding of Blasen or Greeff's cysts in the ciliary epithelium is not constant when the anterior chamber is drained, and neither is it so after the vitreous tappings. However, it was observed frequently and was analogous in every way to the appearances after paracentesis at the limbus. One eye of rabbits 17, 29, 31, 32, and 49 was normal and these were used for comparison. The structure of the vitreous was further studied in eight other

eyes, enucleation having been done following aspiration or vitreous removal, after the following intervals: Twenty-seven minutes in rabbit 49; forty-seven minutes in 51 R. & L. One week in 50 R. & L. One week in 31; twelve days in 18 L. and nineteen days in 18 R.* But five eyes were observed following injection of fluorescin into a vein of the ear, too few from which to draw any conclusions. In only one of these was the vitreous stained after aspiration of vitreous fluid. Also in but one rabbit was there obtained a satisfactory Ehrlich's line following paracentesis of the anterior chamber. It was therefore decided to exclude from consideration the findings after fluorescin injections.

The results of the tests made for albumin in normal vitreous and in vitreous examined subsequent to tapping or aspiration can best be shown in tabular form. Three specimens were sent to the chemical laboratory for examination; the remainder were tested in the eye laboratory, using the heat and cold nitric acid tests. Quantitative analyses were not made, owing to the disorganization in the department as previously mentioned. This should without doubt be done in the future. In this series 22 eyes were examined. In four of the tests the normal fluid was drawn off with a syringe from the living animal. In the remainder the animals were first killed. Ether was found, for this purpose, more satisfactory than chloroform.

It is not intended to convey the idea that the fluid in the normal after testing was perfectly clear and limpid. Such was not the case. But the turbidity after heating or a ring after contact with nitric acid was not sufficient to record it as plus (+). Heat and nitric acid at best are but rough tests, and it is well known that the normal vitreous contains a trace of albumin. The findings in the normal are recorded as a trace (Tr.). In a number of instances the amount of

* Since writing, a number of eyes were examined after intervals varying from three weeks to two months.

TESTS FOR ALBUMEN IN THE VITREOUS.

RABBIT NO.	AMOUNT ASPIRATED *	ENUCLEATED IN—	TEST WITH HEAT	TEST WITH NITRIC ACID
27 O. S.	Not recorded	45 minutes	+	+
O. D.	Normal	45 minutes	Tr.	Tr.
34 O. S.	½ c.c.	55 minutes	+	+
O. D.	Normal	55 minutes	Tr.	Tr.
35 O. S.	½ c.c.	70 minutes	Not made	+
O. D.	Normal	70 minutes	Not made	Tr.
36 O. D.	½ c.c.	50 minutes	+	+
O. S.	Normal	50 minutes	Tr.	Tr.
38 O. D.	½ c.c.	60 minutes	+	+
O. S.	Normal	60 minutes	Tr.	Tr.
39 O. D.	½ c.c.	Living animal	Not made	Tr.
O. S.	½ c.c.	Living animal	Not made	Tr.
40 O. D.	½ c.c.	10 minutes	+	+
O. S.	½ c.c.	10 minutes	+	+
44 O. D.	½ c.c.	Living animal	Not made	Tr.
O. S.	½ c.c.	Living animal	Not made	Tr.
45 O. D.	½ c.c.	30 minutes	Laboratory analysis +	..
O. S.	.4 c.c.	40 minutes	Laboratory analysis +	..
47 O. D.	.4 c.c.	35 minutes	Laboratory analysis +	..
O. S.	Normal	..	Tr.	..
48 O. D.	0.4 c.c.	40 minutes	Not made	+
O. S.	Normal	40 minutes	Laboratory analysis Tr.	..

* It was not the aspirated fluid which was tested, except in rabbits 39 and 44.

vitreous fluid was insufficient to make two tests, so that the nitric acid test alone was made. The most notable changes seen in the ciliary processes of the animals operated upon were—(1) A broadening or thickening of the processes due to engorgement and dilatation of the capillaries. (2) An apparent lengthening with a convoluted formation so that the processes lay in a folded manner against the capsule of the lens. (3) The formation of blisters (Blasen)—the so-called Greeff cysts. (4) A tendency in many specimens for the processes to take on a nodular or beaded appearance. These beaded areas were filled with a faintly staining homogeneous substance. Such staining was likewise observed in other parts of the processes. These areas were presumably filled with a fibrinous fluid. In Greeff's cysts under high power these apparently homogeneous places resolved them-

selves into a most delicate fibrillar network incompletely filling the Blasen, due most probably to contraction and shrinking. In several cysts the center was occupied by one of these delicate networks with fine fibers extending outward in a radial manner to the walls of the cyst. No depigmentation was necessary in albinotic rabbits to demonstrate the nuclei of the individual cells making up the walls of these Blasen, which consisted of a single layer of cells. In other words, the cyst is due to the separation of the two layers of epithelial cells covering the ciliary processes. Occasionally a few red cells were seen within the confines of the blister. The lightly staining homogeneous albuminous substance more or less filling the anterior chamber seldom showed red cells present in those specimens in which a paracentesis of the anterior chamber had been done. Sections were cut in two ways: sagittal and coronal, the latter allowing of the observations of many processes in one section. In the normal the processes were thinner, straighter, often reaching to but seldom touching the lens capsule and never thrown in folds on that membrane. Furthermore, the staining properties were markedly different from those of the animals operated upon. In the normal no Greeff cysts were found. The contention that the so-called cysts might be artefacts is without weight, since they were always found in the experimental animals and not in the normals. In general, at the end of a week, after tapping the vitreous, the ciliary processes had resumed in great measure their normal appearance. Rarely one met with a Greeff cyst after a week, although some broadening of the processes still remained. The characteristic changes were best observed in from one-half to one hour after withdrawing vitreous, which corresponds closely to the finding of the maximum quantity of albumin in the vitreous after tapping. The conclusion cannot be avoided that the same ciliary changes occur after vitreous loss as after aqueous loss, and that in both instances they are solely the result of the

disturbance of the normal ratio between intraocular tension and endovascular pressure.

Attention has been directed to the fact that preserving fluids and reagents may effect the delicate fibrillar network of the vitreous to the extent of producing a dense feltwork, at least denser than the normal fibrillar structure is normally thought to be. The findings noted in the vitreous structure in the main were not satisfactory. Nothing was found to disprove the statement of a number of writers that the vitreous as vitreous does not regenerate. The inherent difficulties in a study of the vitreous fibrillar structure are enormous. Fixation and hardening, even when carefully and slowly done, are attended with so much shrinking that it is difficult to draw definite conclusions. This is true of the normal and more so of the experimental cases. Withdrawal of more than 0.5 c.c. of vitreous fluid is often accompanied by hemorrhage and retinal detachment, which introduce disturbing factors in an experimental study of the vitreous. In all the specimens, toward the retina, a broad area of parallel, wavy, minute fibrils, not interlacing, was observed. This could not be considered as constituting a capsule as the area was much too wide. It is more logical to assume that it was the result of the fixing fluids. Quite sharply demarcated from this toward the lens were the typical interlacing, closely aggregated fibrils of the fibrillar structure. In places these fibrils appeared broken off or fragmented, with spaces between very much larger than in the normal, in which they are exceedingly minute. Nothing was seen to suggest a division of the fibers or any attempt at a restoration of the fibrillary structure.

SUMMARY.

1. The fluid filling the anterior and posterior chambers and the meshes of the vitreous fibrillar network is aqueous fluid and is derived from a common source, that source being in all likelihood the epithelium of the ciliary processes.

2. The fluid exchange of the vitreous and aqueous humor is exceedingly slow under normal conditions, and the quantity of aqueous continuously formed is obviously very small. When, however, normal conditions are disturbed, as by sudden lowering of intraocular tension, the genetic apparatus is so demoralized that a modified serum passes over into the aqueous, the selective and differentiating activity of the ciliary epithelium being for the time abrogated.

3. The presence of increased albumin can be demonstrated in the vitreous fluid following aspiration or vitreous decompression analogous to the phenomenon occurring after drainage of the anterior chamber.

4. The changes in the ciliary processes are similar, if not identical, in the two procedures.

5. It is fair to conclude that such changes are the result solely of the disturbance in ratio between intraocular and endovascular tension, a product of disturbed physiology.

6. The fibrillar structure of the vitreous once destroyed or lost is not replaced or regenerated.

7. The increased albumin content following vitreous decompression is slow in disappearing, a trace (slightly greater than the normal) being demonstrable after twelve days. The changes in the ciliary processes recede more rapidly, and the processes are virtually restored to normal after a week

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THE THERMOPHORE IN OPHTHALMIC PRACTICE.*

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The thermophore for ophthalmic use is an instrument evolved by the writer through a series of studies on the "Effects of Heat on the Eye," extending through a period of several years. Its function is to conduct *measured* intensities of heat directly into the eye tissues through metallic masses placed in actual close contact with these tissues.

Heat has long been used in various eye affections, as hot applications to the lids, steam or hot-air jets against the cornea, red hot wires or metallic points in contact with an ulcer, or hot masses of metal held near a corneal ulcer; but in none of these methods was there any means of regulating the intensity of the heat applied or of knowing how much heat actually entered the tissues or how great an intensity of heat it took to meet any given pathologic condition. The effect has been largely chaos, and considerable disappointment in results.

The thermophore was designed to overcome these defects, and has already produced results of marked importance in ophthalmic practice.

It is well known that different materials have different degrees of conductivity for heat as well as for electricity. If, for instance, a piece of glass and a piece of brass are each heated to 170° F. and held against a rabbit's cornea for one minute, the brass will produce complete destruction of the corneal epithelium, and intense clouding of Bowman's

*Candidate's thesis for membership accepted by the Committee on Theses.

membrane and the substantia propria, while the glass will produce no effect whatever. It is essential, therefore, to select some convenient material and stick to it throughout all our experiments and practice. The heat conductivity of brass is not as high as that of silver or copper or gold; but brass is much easier to get commercially in convenient forms than gold or silver, and much easier to work than copper. Accordingly brass, plated with nickel, was the material chosen, and the method was invariably to hold the metallic masses, shaped to convenient forms and denominated "applicators," directly against the tissue to be heated so that not the smallest air gap intervened. If any sort of air gap is allowed to intervene, none of the effects hereafter described will take place.

After selecting a suitable conductor the next thing necessary is some convenient and reasonably accurate means of measuring the varying intensities of heat to which the applicators are raised. The most generally used instrument for this is the mercury thermometer, and that is the instrument adopted for all clinical purposes in this work. The applicators are made of short pieces of brass rod, the centers of which are bored out so as to receive the bulb of the thermometer in such a way as to place the mercury very near the contact tip.

This gives a good method of measuring the average temperature of the metallic mass, but does not measure the exact temperature of that part of the surface placed in contact with the tissue to be heated. Moreover, this method is entirely unsuitable for measuring temperatures in small spaces such as the anterior chamber. For this purpose resort must be had to the thermo-couple. This method of measuring temperatures in the eye was first used by Prof. Michel,* but my attention was called to it by a paper of

* Michel: Die Temperatur-Topographie des Auges, Arch. für Ophth., xxxii, 1, 227–238 (1886).

Dr. Lucien Howe.* It depends upon the principle that when two dissimilar metals, as iron and silver, are soldered together in three pieces so as to form two junctions, a flow of electric current is set up if there is a difference in temperature of the junctions. Now if one of the junctions is held at a constant temperature, the flow of current will depend solely on the temperature of the other junction. Galvanometers are constructed so as to measure small quantities of electric current with great accuracy. A galvanometer provided with a telescope and graduated scale can be placed in such a circuit and the temperature of one of the junctions varied by definite amounts with the aid of a resistance coil and thermostat until a definite relationship is established between the temperature of the junction and the scale of the galvanometer.

This relationship is plotted on coördinate paper and held for reference. The dissimilar metals can be used in the form of fine wires insulated by being painted with celluloid varnish. One of the junctions can be kept at a constant temperature for several days at a time by immersing it in a vacuum bottle filled with cracked ice and water. When it is desired to ascertain the temperature of some part of the anterior chamber, for instance, and our metals are iron-silver-iron, we attach the free end of one wire to a pole of the galvanometer, place the iron-silver junction in a vacuum bottle, thread the free end of the other iron wire through a small surgical needle and draw the wire through the anterior chamber until the silver-iron junction occupies the part of the anterior chamber the temperature of which is desired. The needle is then removed, and the free end of the iron wire is attached to the other pole of the galvanometer. There will then occur a definite swing of the galvanometer needle corresponding to a definite temperature of the junction in

* Howe: Temperature of the Conjunctiva, Journal Amer. Med. Assn., 1913.

the anterior chamber. This temperature can be read off from the chart constructed as above.

It takes some weeks of painstaking labor to construct such a chart, and constant checks must be employed during its use to prevent misleading results. While, therefore, it is of great value in the laboratory, it is not used for clinical purposes.

Having selected our conductor and our means of measuring temperature, it is now necessary to devise some way of varying the temperature of our metallic masses.

The most primitive way, and an effective one, is to heat the masses directly by holding them in a flame. A more convenient, and from several standpoints a better, way is to wrap a sheet of mica about the mass, then wind a piece of resistance wire around it and raise the temperature by allowing an electric current to flow through the wire.

In fact, it is best not to heat the metallic masses (applicators) at all, but to provide a tube within which the applicator snugly fits and apply the heat to the tube. This provides an easy means of varying the shape of the tips of the applicators to meet varying conditions.

Finally, it is necessary to provide some method of heat insulation. This is done by the use of felt or friction tape and rubber tubing about the tube or core within which the applicators fit.

The first completed thermophore was shown in June, 1916.* It was designed for keeping the applicators at a constant temperature for indefinite lengths of time.

The heat was generated in it by means of a resistance coil and kept constant at any desired point by means of a platinum tipped thermostat. This instrument required some care in its use, and is not now employed for clinical purposes, but certain important determinations were made with it.

The first use to which it was put was the determination of

* Ophthalmic Section, Amer. Med. Assn., Detroit.

the physiologic limits of tolerance of different tissues of the cornea for the conductor used (nickel-plated brass) for contacts of ten minutes' duration. The corneæ of rabbits were used for this purpose under local anesthesia (cocain 5 per cent., adrenalin $\frac{1}{2000}$) or general anesthesia (ether).

It was found that up to 116° F. (46.6° C.) no effect was noted on the eye, except a marked miosis; that changes in the corneal epithelium began to be noticed at temperatures above this, and that at 120° F. (48.0° C.) complete destruction of the corneal epithelium occurred over the whole area heated. Unless very large areas were destroyed, the epithelium rapidly replaced itself without any perceptible damage to the eye; 124° F. (51.1° C.) caused temporary clouding of the substantia propria; 128.4° F. (53° C.) caused more prolonged clouding of the substantia propria with subsequent clearing and gray atrophy of the iris with closure of the filtration angle opposite the point of application. This local closure of the filtration angle became visible about two weeks after the heat application when all signs of inflammation had ceased, and is suggestive of interesting experimental possibilities. At 135.3° F. (58.4° C.) occurred marked immediate clouding and subsequent softening of the substantia propria, leading to conical bulging with subsequent nearly complete clearing.

By means of a thermo-couple junction, placed within a hole drilled through the applicator point as near as possible to the surface in contact with the eye, it was found that beyond 125° F. (85° C.) the temperature of the surface in contact with the cornea, while in contact with the cornea, lagged somewhat behind that indicated by the thermometer. At 131° F. the surface was 2° behind the thermometer and at 135° F., 3.6° behind. In the laboratory experiments corrections were made for this lag, but in clinical work no account whatever is taken of it.

After these facts had been determined applications of

various degrees of heat for ten-minute intervals were made on an important pathologic condition: serpiginous ulcer of the cornea, hypopyon keratitis. A virulent strain of pneumococcus was obtained and attempts made to inoculate it into the cornea of rabbits' eyes. It was found insufficient to merely remove an area of epithelium and place the organisms upon the denuded area, but by the use of a triangular needle, the surface of which had been roughened by oxidation, Bowman's membrane was broken through and the organisms inserted into the substantia propria. When this technic was adopted a virulent ulcer was obtained on every attempt. Some of these ulcers were accompanied by hypopyon that half filled the anterior chamber, while others were not accompanied by hypopyon at all. There was always a tendency to spontaneous healing, so that each eye of each rabbit was inoculated. One eye was treated by varying degrees of heat up to 130° F. (54.4° C.) for ten-minute periods, and its progress compared with that of the opposite eye.

The net therapeutic result of this technic was total disappointment, and attention was turned to another method which was reported in June, 1917.* This consisted in the short application of gradually increasing degrees of heat to pneumococcous ulcers produced as above until a point was reached where the organisms were definitely killed within the substantia propria. The duration of application was limited to one minute, because this is about the time it takes for the full effect of heat to pass through the whole thickness of the cornea, as shown by thermo-couple measurements. The minimum intracorneal death-point for pneumococci was found to be about 152° F. (66.6° C.). Proof that this point had been attained rested upon two factors:

1. Rapid cessation of clinical symptoms as compared to similar symptoms in the opposite eye used as a control.

* Ophthalmic Section, Amer. Med. Assn., New York.

2. Sterile cultures after the application had been made.

With the adoption of an application of one minute's duration, the use of a simplified instrument was possible, and such an instrument was the one shown. It consisted of a core reamed out at one end for receiving variously shaped applicators and fitted at the other end with a lock-nut or piece of steam packing for the reception of a thermometer; also a felt-lined jacket for retaining the heat in the core. To use this instrument an applicator is selected with a point just large enough to completely cover the ulcer, or a selected part of the ulcer, to be treated. The thermometer is then slipped into the core until its bulb rests within the hollow of the applicator, and, using the thermometer as a stem, the core is held over a flame so that the flame plays along its middle (not on the applicator) until the mercury in the thermometer rises to a point above that at which the applicator is to be used on the ulcer. The core is then slid into the felt-lined insulating jacket, and the mercury observed until it falls to the desired point, at which instant the applicator is held against the ulcer for one minute.

About fifty cases of hypopyon keratitis have been treated in human eyes by colleagues, associates, and myself, in St. Louis, by means of this instrument, and a number of cases by Dr. E. S. Thomson, of New York. It has been found, as a result of experience, that it is best to apply the points at 158° F. or in severe cases 160° F. (70 to 71.1° C.) in order to terminate the process by one application of one minute's duration. The temperature is, of course, constantly falling in the instrument, and, by the end of the one-minute period, the thermometer may indicate 150° F. (65.5° C.), or even lower. The results have been so uniformly successful that they justify a reasonable hope that the method is specific for this particular trouble. The technic is, briefly, as follows:

1. Avoid the use of the words *burn* or *heat* in the presence

of the patient. These are terrifying words in connection with so sensitive an organ as the eye, and some partial failures resulted in our cases because of the nervousness resulting from them. If any explanation at all is necessary, state that just enough *warmth* is applied to the ulcer to stop the poison without hurting the eye.

2. Anesthetize the eye *thoroughly* with a mixture of cocain, 5 per cent., in adrenalin $\frac{1}{4000}$. While this is being done several applicators may be tried on the ulcer so as to select one that will just cover it. After the selection of a suitable applicator, the applicator may be slipped into the core, the thermometer put into position, and the core slid into the insulating jacket, as if ready for use. Then the point (still cold) is again applied to the ulcer so as to select the best object of fixation for the patient, and the best light in which to view the eye; also, incidentally, to acquaint the patient with the instrument and disassociate it in his mind from pain.

3. When the eye is thoroughly anesthetized, withdraw the core from the insulating case and hold it over a flame, allowing the flame to play along the middle of the core and not on the applicator. It is just as well to do this out of sight of the patient. When the mercury has risen above 158° F. (70° C.), slide the core back into the insulating case. Lay the instrument on a table or somewhere in the patient's sight and instil another drop or two of anesthetic.

4. As the mercury nears 158° F. (70° C.) pick up the instrument, have the patient's lids held by an assistant or with your free hand, or insert a speculum. The moment the mercury reaches 158° F. (70° C.) or, in fact, just after it falls below 160° F. (about 71° C.), place the point of the applicator in *direct contact* with the ulcer, and keep it gently pressed there for one minute. If the anesthesia has been satisfactory, and the patient's lids are not allowed to touch any part of the applicator, there will be little or no pain

experienced. Many of our patients could not perceive any difference between the final application and the preliminary cold one.

5. Use some mild cleansing agent, as argyrol 25 per cent., and irrigate with normal salt solution. Atropin must be used according to indications for iritis, which is always present if the stage of hypopyon has been reached.

After Treatment.—For a few hours there will be pain, beginning about one-half hour after the application. This is a direct result of the heat, and can be controlled by the use of cocain or cocain and atropin. No other treatment is necessary. Next day the physician will almost invariably be in doubt as to whether the ulcer is improved or not. The hypopyon will be unchanged, or somewhat larger. The ulcer will seem larger because of the destruction of the epithelium around the margin of the ulcer. There is, however, in a majority of cases, one strikingly favorable feature: the patient will have had a comfortable night, and the eye will be free from pain. Treatment will be fluorescin, 2 per cent., to demark clearly the limits of denudation of Bowman's membrane, argyrol as a cleaning agent and as a stain marking the areas where Bowman's membrane is broken through. Normal salt irrigation and atropin as needed. This should be the daily treatment until the case is discharged, unless there is an exacerbation.

On the second day there should still be freedom from pain and general improvement. The hypopyon should be diminishing and the epithelium growing in from all sides. This should continue from day to day until cure is established. If an exacerbation occurs, it will be anywhere from the second to the sixth day. In such an event another, and in very severe cases a third or fourth, application may be necessary.

Causes of Failure.—If the ulcer is larger than the surface of the tip of the applicator selected, and the physician

attempts to make up for this by moving the point about over the ulcer during the time of the application, the heat will be so diluted as to be ineffective, and the ulcer will be growing more vigorously next day than on the day of the application. The same result may come about from a nervous person rolling his eye about so as to repeatedly interrupt the application. In such a case it is necessary to prolong the application to make up for the time lost. If necessary, the instrument must be reheated and reapplied.

The ulcer may be large and irregular in shape. In this case several applicators may be used, each one on a different part of the ulcer continuously for one minute until the whole ulcer has received the full exposure of one minute with an initial temperature of 158° F. (70° C.). More or less overlapping of the applications does no harm. In such cases there is usually no hope of much or any vision, and the whole object is to get the patient comfortable and save a globe that will give him no further trouble.

Perforation occurred in a number of cases. This was almost always of pin-point size and in the deepest part of the ulcer. The iris was rarely engaged, and recovery appeared to be hastened rather than retarded by it.

Enucleation was never necessary. Several cases were complicated by chronic purulent dacryocystitis. These did not show any special liability to reinfection, much to our surprise, and ran a course as favorable as the simpler cases. The general corneal leukocytosis following in a day or two after the application probably explains this.

The mode of action of the heat is as follows:

1. Direct destruction of the organisms in the tissues by the intensity of the heat used. This is about the same as is required for the immediate destruction of pneumococci in cultures. The toxins are also much diminished in virulence by this degree of heat. This probably accounts for the rapid, and in most cases, complete relief from pain.

2. The induction of a rather marked exudation from the area heated shortly after the heat has been applied. This causes a considerable discharge of pus cells, leukocytes, bacteria, etc., which sometimes leaves the ulcer remarkably clean and clear within a few hours.

3. The induction of a noticeable leukocytosis of the whole cornea. This begins a day or two after the heat has been applied and probably has much to do with the rarity of reinfection.

The success attending the use of the thermophore in hypopyon keratitis suggests the possibility of it also being useful in other conditions. Dr. Ewing used it on the rough areas in a case of chronic trachoma at 143° F. (61.7° C.) for one-half minute. This resulted in an hour or so in the desquamation of a pellicle of epithelium, somewhat larger than the area heated, without any bleeding from the surface. The areas healed by fine granulation, and the patient reported much improvement in the feeling of roughness, of which he had been complaining.

Dr. Hardy and Dr. Wiener have used it in non-pneumococcous ulcers with prompt relief and recovery.

Dr. E. S. Thomson has used it in a case of disciform ulcer with "prompt result."

The cornea, and for most part the sclera, is largely composed of collagenous material, which has a temperature resistance distinctly higher than more succulent structures containing blood-vessels, etc. On this basis there are still possibilities not yet determined, and which can only be worked out as clinical opportunities present themselves.

No opportunity has yet presented itself of trying it, for instance, on a case of a corneal or corneo-scleral epithelioma. Also, it has not been tried out in dendritic or bulbous keratitis, nor on gonorrheal ulcers. These possibilities in the extension of its use, as well as more convenient use in hypopyon keratitis, warrant study in improvement in several

directions. If the instrument is smaller than either of those described above, it will be advantageous in allowing a better view of the eye being treated. If the applicators are heated by simply pressing a button in the side of the instrument, time will be saved and the patient will not think of his eye as being "burnt," but will probably conclude that some sort of electrical treatment is being used. The thermometer should be long enough for readings of 110° F. (43.3° C.) to 180° F. (82° C.) to be easily made, and the instrument should be so constructed that the temperature may be held about any desired point as long as necessary. It should be as simple as possible to avoid getting out of order. Several such instruments have been constructed and tried out in laboratory tests. They consist of a central tube which is long enough to hold the thermometer fixed in position; in fact, it is somewhat longer than the thermometer which is sealed in it and protected by it throughout its whole length. One end of the tube is reamed out to hold the applicators which are much less in general bulk than those used in the instruments described above. About this end is wrapped a sheet of mica, and about this is wound four feet of resistance wire of which the resistance is about five ohms per foot. The resistance wire is bound down by friction tape. Just back of the heat coil are two pieces of heavy brushing brass, one of which has screwed to it a piece of cylindric fiber to act as a push button for making and breaking the current.

All this has over it a piece of plated tubing so that nothing shows but the push button at the side of the tubing. The wire from the push button goes directly to the plug; that from the heat coil to another resistance coil consisting of 16 feet of resistance wire with total resistance of about 80 ohms. The wire then goes from this coil to the plug. This arrangement serves for a 110-volt alternating current. Experiments are now under way for choosing sufficient resistance to serve either with the direct or alternating current.

If the instrument arranged as above is used in 110-volt direct current, the amperage will be so large as to burn the contact points at the push buttons. With the alternating current if the resistance wire is wound in spirals parallel to one another there are induced currents set up which have a "choking" effect on the current to the thermophore, so that less is delivered than in the case of 110-volt direct current.

With a little practice the temperature of the applicator can be kept constant by occasionally pressing the button in the side of the instrument with the index finger as long as required without removing the instrument from the area being heated. Since this instrument is intended to be used in various pathologic conditions besides that of hypopyon keratitis, it is well to define its action at various temperatures on the cornea and conjunctiva of rabbits' eyes by actual experiment. It is found that with an applicator four mm. in diameter at the tip, an application for one minute at 120° F. (48.8° C.) to the cornea caused very slight changes in the epithelium, marked by faint immediate staining with fluorescin; all trace had disappeared two days later; 125° F. (51.6° C.) caused more marked immediate staining, all trace of which had disappeared in two days; 130° F. (54.4° C.) marked general immediate staining over the whole area treated, complete replacement of epithelium in two days with a faint opalescence remaining over the area; 135° F. (57° C.) applied to the conjunctiva of the rabbits' third lid caused marked immediate hyperemia without hemorrhage. Two days later there was still marked hyperemia with small hemorrhages scattered about through the area heated. These effects of varying degrees of heat on different kinds of tissues must be considered in the use of the thermophore in pathologic conditions for which it has not yet been accurately classified.

The new form of the instrument cannot be manufactured for general use until after the war.

THE PRESENT STATUS OF DENTAL SEPSIS IN ITS RELATION TO CERTAIN OCULAR DISORDERS.*

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That focal infection plays an important part in the development of systemic or general disease is a well-established fact. The brilliant work of Billings, Rosenow, and others, in a series of clinical and laboratory investigations, has furnished incontestable proof that many of our former conceptions with regard to the etiology of disease have been fundamentally wrong.

Older observers long ago pointed out that infectious disease was the result of bacterial invasion, and modern bacteriologists and clinicians are today giving us conclusive evidence that many of the systemic diseases, heretofore classed as toxic, metabolic, or nutritional, are due to virulent microörganisms set free from a focus of infection. Not only has it been shown that many systemic diseases are the result of bacterial invasion but that disease in special organs frequently originates from focal infection.

Ophthalmologists have not been slow to recognize the very intimate relationship between focal infections and certain diseases of the eye whose etiology had either remained obscure or had been based upon erroneous conceptions. Clinical research, laboratory investigation, and animal experimentation have demonstrated beyond question that a number of ocular lesions can be directly traceable to septic foci in the teeth, tonsils, nasal accessory sinuses, and even in more remote parts of the body.

* Candidate's thesis for membership accepted by the Committee on Theses.

That dental disease, and particularly suppurative processes, can be productive of ocular disturbances is a fact that has been established for many years. Even older writers pointed out that a relationship existed between dental caries and certain ocular manifestations. It has remained, however, for modern observers, both in ophthalmology and odontology, to clearly define this relationship and to establish indisputable evidence of the baneful influence of dental sepsis in the production of certain pathologic states of the eye.

The marked development in dental *x*-ray work has certainly, to a great degree, been the means by which it has been possible to demonstrate certain dental lesions whose presence would have been otherwise unsuspected and too often undetected. In most instances it is just such lesions which have been proved to be the responsible factors in the development of secondary eye conditions.

Coincident with the progress of dental roentgenology has been the gradual recognition of the fact, on the part of dentists, that, while the ability to do good mechanical work is a desirable asset, the elimination of sepsis is the goal toward which their best efforts should be directed. This, together with a willingness to coöperate with ophthalmologists, has brought about a more exact knowledge of the subject than would otherwise have been obtainable.

While the literature has been quite voluminous during the past two years, and has been replete with many instances and case histories in which ocular lesions could be more or less directly traceable to dental sepsis, cognizance should be taken of the fact that, despite very earnest efforts on the part of many active workers in ophthalmology to ascertain the true cause of any given disease, in the enthusiasm of the moment we are sometimes apt to draw conclusions which may undergo extensive alteration after more mature deliberation.

As Zimmerman* very strikingly states, "We may expect to read in our literature for some time to come reports of so-called ocular inflammations dependent on dental infections. Such infections will have a 'boom' for a while; but, as Dr. Herman Knapp once said, 'We cannot reach a satisfactory conclusion on any subject until its 'boom' has subsided.'" Then we will probably realize that such infections do play a rôle, but not nearly so important a rôle as seemed to be the case during the "boom."

While it may be admitted that we are passing through this stage in the study of the relationship between dental infections and ocular disorders, sufficient evidence has been submitted, in the opinion of the writer, to justify the conclusion that oral septic conditions play an important part in the development of certain eye lesions and we are now able to make logical deductions as to their influence.

Quoting from an article by Arthur D. Black,† it may be said: "From the viewpoint of dental pathology, the ocular disturbances which result from dental lesions should be divided into two groups: (1) Reflex neuroses, presenting as sensory, vasomotor or muscular disorders; (2) inflammation of the various tissues of the eye, due to infections. The former are practically always due to irritation of a vital pulp of a tooth, although an occasional case may be due to an impacted tooth; the latter are practically always due to an infection involving the peridental membrane, either in the form of an alveolar abscess at the root apex, or a pus pocket alongside the root."

The same author‡ further makes this significant statement: "It is safe to say that fully 50 per cent. of persons twenty-five years old have infections which have destroyed areas of bone about the teeth sufficiently to be easily de-

* Ophthalmic Year Book, vol. xiii, p. 136.
† Ophthalmic Record, vol. xxiv, p. 610.
‡ Dental Summary, vol. xxxvii, p. 714.

tected by roentgenoscopy, and that this percentage gradually increases until it approaches 100 per cent. in persons who are not edentulous at fifty years of age. The estimate made several years ago by Dr. T. L. Gilmer that 75 per cent. of adults would be shown to have chronic infections involving the maxillary bones, is fully supported by statistics thus far gathered."

While it is known that the mouth is the habitat of many virulent microörganisms, particularly of the streptococcus and pneumococcus types, and while the very great prevalence of dental sepsis would seem to justify the statement made by some writers that the mouth is the most frequent offender in cases of secondary eye lesions, it must be constantly borne in mind, as Billings* points out, that "the natural defenses of the body, due to bacterial and antitoxic powers of the tissues, blood plasma and cells, especially the phagocytes, protect the majority of us from the acute infectious diseases."

It is, therefore, not unreasonable to assume that nature furnishes the same effective barriers in her effort to protect special organs from the invading bacteria originating from a septic focus.

With regard to immunity, Billings further states: "All individuals do not possess an equal degree of natural immunity; some more readily succumb to the invading infectious agents. When the animal body is invaded with pathogenic bacteria the natural defenses are increased by their presence in the tissues and blood. These processes are: (1) The phenomenon of positive chemotaxis with resulting leukocytosis and the accumulation of leukocytes in the areas of infection by the formation of local exudates, liquid (purulent) and fibrinoplastic, which may serve as walls of protection against further direct invasion; (2) leukocytic phagocytosis with destruction of the invading bacteria;

* "Focal Infection," Billings, p. 16.

and (3) the formation of protective anti-bodies in the blood and tissues."

The development of these protective processes, in nature's effort to rid the body of disease, certainly offers a satisfactory explanation of the immunity which certain individuals acquire both in systemic and local disease. It further explains the fact that, though a large number of individuals are the victims of dental sepsis, a comparatively small number suffer from secondary eye lesions because of its presence. On the other hand, it can be definitely asserted that a greater susceptibility to systemic or local disease from focal infections exists in some persons than others. Innumerable instances can be cited in which the body resistance is so lowered because of environment, habit, modes of living and many other influences that a successful bacterial invasion takes place and disease is the inevitable result.

Again, it may be noted that bacteria of a local infection may attain a specific pathogenic and elective tissue affinity. The clinical observation of Billings and his co-workers, together with the numerous animal experiments and rational deductions made by Rosenow, have confirmed the accuracy of this principle, and have emphasized its importance in the study of focal infections. Such focal infections in the language of Rosenow,* quoted by de Schweinitz, "are no longer to be looked upon merely as a place of entrance of bacteria, but as a place where conditions are favorable for them to acquire the properties which give them a wide range of affinities for various structures."

On the other hand, some investigators, particularly Hartzell, and Henrici, take issue with Rosenow's elective localization theory, and have found no evidence in their own experiments to confirm it.

In a very exhaustive article on "The Pathogenicity of

* Ophthalmic Record, vol. xxiv, p. 602.

Mouth Streptococci," these authors,* in referring to the relationship between peridental infections and systemic disease, state that they do not believe that mouth streptococci have been proved to cause any other systemic disease than the various lesions that go to make up the disease known as rheumatism. While the influence of dental sepsis in certain ocular manifestations is not mentioned in this article, it may be inferred, from the reference of the authors to so-called rheumatic affections, that iritis might be included in the list of infections due to septic foci originating in the teeth.

The bacteriologic relationship between diseases of the mouth and eye infections has not received the attention that one might expect would be accorded it in the numerous papers devoted to the study of ocular disorders and dental sepsis.

Leyy, Steinbugler, and Pease,† in a study of 57 cases in the Herman Knapp Memorial Hospital, record the bacteriologic findings in a few of the cases reported. Cultures obtained from the offending teeth in most instances yielded the streptococcus viridans as the predominating organism. In a few cases staphylococci and pneumococci were obtained.

It is interesting to note that vaccines were obtained and used in the treatment of some of these cases, and while the eye condition cleared the evidence is not of sufficient weight to prove that the use of the vaccines, after removal of the primary focus of infection, influenced appreciably the rapid recoveries reported. Certainly in the light of our present experience, notwithstanding some reports to the contrary, vaccine therapy has proved disappointing in this class of cases, and no evidence has yet been submitted, in the opinion of the writer, which would seem to justify any other conclusion.

* Jour. National Dental Ass'n, vol. iv, No. 5.

† Dental Summary, vol. xxxvii, No. 10.

In this connection may be mentioned the case of toxic choroiditis secondary to chronic alveolar abscess reported by Rowe* and treated with vaccine therapy. In this interesting communication the author describes the case history of a patient whose right eye became the seat of an active choroiditis with a densely infiltrated vitreous and deposits in Descemet's membrane. The vision was reduced to counting fingers at ten feet. The etiologic factors proved to be two central upper incisors above which was a large, badly necrotic area of bone. Extraction of these teeth with removal of the necrosed bone was followed by rapid improvement in sight and general health. In ten days from time of operation the vision had risen to 20/49, deposits in Descemet's membrane had lessened, the vitreous opacities were less numerous, and the nerve-head and eye-ground were quite visible. No treatment, other than the local use of atropin and dionin, had been used up to this time. The administration of mixed infection phylacogens was then begun and continued in minute doses for a period of one month, at the end of which time the eye appeared to be normal.

The question that very naturally presents itself in the conduct of this case is, why was it necessary to institute a vaccine treatment in a patient whose eye had shown such rapid improvement after removal of the primary source of infection? The fact that the vision rose from ability to count fingers at ten feet to 20/49 in ten days, and that the inflammatory symptoms were rapidly subsiding, would certainly seem to indicate that nature's forces were fulfilling their mission. Rowe's case is both interesting and instructive, but the writer cannot feel that the happy result he obtained was due to the use of a stock vaccine, as might be inferred from the title of the article.

Claiborne's† three cases, two of mild anterior uveitis and one of choked disc, in which the complement-fixation test

* Arch. of Ophth., vol. xlvi, No. 3. † Ophth. Sec., N. Y. Acad. Med.

for the streptococcus viridans was positive, and which were treated by vaccines from stock cultures, might serve, in some measure, to stimulate confidence in vaccine therapy. It should be noted, however, that in these cases no focus of infection or other causes were found to account for the inflammatory condition, and the administration of vaccines was undertaken solely because the complement-fixation test was positive.

In the discussion which followed, Levy and Dwyer stated that their experience had been at variance with the findings of Claiborne. Levy thinks that dental infection is the primary source of the streptococcus viridans in the majority of these cases, and that when the germ enters the blood it becomes a matter of serious import. Dwyer further records that his results with streptococcus viridans had been absolute failures, and that he does not regard the complement-fixation test as reliable in comparison with blood cultures. He likewise lays stress on the fact that vaccines are of little value unless the focus of infection is found and removed.

In the study of the cases which are to follow and which were selected from his private records for the past year, the writer claims no originality for any views he may present or deductions he may make. His desire has been to make an impartial survey of the situation, to glean from the literature such facts as seemed to have an important bearing on the subject, and to base his conclusions upon the findings of others and the experience he has gained from the observation of a limited number of cases.

In tabulating the cases, it has seemed wise not to include any condition which may have been the result of a mixed infection. The object has been to record only those cases in which dental sepsis has been the exclusive factor. As far as possible other factors have been eliminated by the usual diagnostic methods, though it is perfectly possible that in

some instances, and particularly in the cases in which no improvement is recorded, these may have been overlooked.

The following classification of 25 cases tabulates the results, which the writer wishes to record, for the past year:

Diagnosis	Cases	Cured	Improved	Not Improved
Acute iritis	4	4	0	0
Chronic iridocyclitis	3	2	1	0
Suppurative choroiditis	1	0	0	1
Iridochoroiditis	1	0	1	0
Choroiditis	3	2	1	0
Chorioretinitis	2	1	0	1
Scleritis	2	2	0	0
Paralysis accommodation	2	1	0	1
Accommodative asthenopia	2	1	0	1
Retinal hemorrhage	2	2	0	0
Neuroparalytic keratitis	1	0	0	1
Optic neuritis	1	1	0	0
Optic atrophy	1	0	0	1
Total	25	16	3	6

It is a significant fact that in this series the uveal tract has been much more frequently involved than any other parts of the eye. In 18 of the 25 cases the choroid, the ciliary body, and iris have been the structures which have been affected as the result of septic foci in the teeth or of reflex irritation originating in the dental region. This experience is in accord with the findings of Levy, Steinbugler, and Pease in the larger series of cases reported, and is not at variance with the experience of other observers, who have thus far recorded case histories.

Rosenow has noted iridocyclitis in a number of animals, chiefly rabbits, following the intravenous injections of streptococci and other organisms from various lesions of man and animals. Brown, Irons, and Nadler,* in an experimental study of iridocyclitis in rabbits, have also demonstrated the presence of streptococci in the ciliary body

* Arch. Ophth., vol. xlv, p. 229.

and anterior chamber from intravenous injections of cultures obtained from an infected lacrimal sac.

Worth states that more than 50 per cent. of cases of iritis are due to oral sepsis and kindred conditions, while B. F. Lang, out of 176 cases of iritis, found that 71 could be traced to mouth infections. William Lang reports that out of 215 cases in private patients, attributed to sepsis, 139 were traced to pyorrhea or other dental conditions, and he likewise records the interesting fact that in this series the iris was affected 87 times, the ciliary body 79, and the choroid 65 times. De Schweinitz and others have emphasized the fact that general oral sepsis or extensive pyorrhœa alveolaris need not necessarily be present as the instigator of uveitis. It is pointed out that the infection is often due to a small tooth root abscess, which is not discoverable until the *x*-rays are employed.

In fully 75 per cent. of his cases the writer can attest to the accuracy of the foregoing view. Blind dental abscesses were the etiologic factors in this percentage, and in the majority of cases the patients were unconscious of their presence and were loath to believe that a septic process could exist without local irritation or pain.

Another fact which has been brought out prominently in the study of these cases is that even in the presence of extensive pyorrhea, which has existed for many years without causing trouble, the sudden development of an apical abscess will frequently prove to be the exciting cause of the ocular lesion, rather than the pathologic process in the gums.

This emphasizes the importance of a complete *x*-ray examination in even those cases in which the presence of pyorrhœa alveolaris has been definitely established and might be regarded as the only factor in the development of secondary lesions in the eye.

A case history, very much condensed, of one of the writer's

patients, will serve to illustrate the accuracy of the foregoing conclusion:

Mrs. W. F. R., aged fifty-five years, complained of dimness of vision and "spots" in the left eye. She stated that a year previous to her visit she had been under the care of an eminent internist in a neighboring city for some constitutional trouble, the nature of which she did not know. She underwent a number of expert examinations and was subjected to every known diagnostic test.

Her malady, whatever it may have been, failed to clear up until she had visited a dentist, who discovered a well-marked case of pyorrhœa alveolaris, the elimination of which restored her to health. She was sent home and referred to a local dentist with instructions to have her gums examined at stated intervals.

An examination of the eye disclosed a beginning uveitis, with decided infiltration of the vitreous and some secondary iritis. Her dentist, a very competent man, assured the writer that no local infection, other than very slight traces of pyorrhea, then existed and that this was rapidly clearing. Despite the active use of the usual constitutional and local remedies the condition grew rapidly worse. Exudates appeared on the posterior surface of the cornea, the iris became agglutinated to the capsule of the lens by several firm adhesions, and the vision was much reduced.

While her teeth had been *x*-rayed before with negative results, it was determined to make another search for focal infections. The film disclosed an apical abscess involving one of the bicuspids. The tooth was extracted, the abscess drained, and in a comparatively short time the eye was quiet and regained normal vision.

The hidden element of danger lurking in so many crowned teeth can certainly be regarded as an exciting factor in many cases in which the ocular structure is involved from dental sepsis. In the writer's cases in several instances it was apparent that crowned roots or roots covered by bridge work were the responsible factors in the production of the eye lesion.

The principle of building up an old root, and in many cases a septic root, is, of course, essentially wrong, and it is inconceivable, in the light of our present knowledge, why this practice should be continued. Dental roentgenology will in a great measure serve to minimize this evil in the future, but until it is universally adopted the crowned tooth will continue to be one of the principal sources of focal infection in the dental region.

As an example of its potent influence in the production of ocular lesions may be cited the case history of a patient in the writer's series:

A. W. W., aged thirty-eight years, a perfectly sound and healthy looking man, had been under the care of the writer for a period of four years for recurrent attacks of iritis in the left eye. The inflammatory exacerbations were always of the most severe type, being accompanied by fibrinous exudates in the anterior chamber and numerous pupillary adhesions.

The patient gave a history of an occasional attack of muscular rheumatism, but otherwise had always been healthy. Every region of his body, except the dental region, had been explored in the hope of locating the source of his infection. Syphilis, tuberculosis, and gonorrhea were eliminated, and the nasal accessory sinuses showed no signs of involvement.

Failing to find any other responsible factor, the case was finally recorded as "rheumatic iritis," a term which has been useful in bygone days in describing a doubtful etiology. Physiologic doses of the salicylates were administered, and the usual local measures instituted. Despite very energetic treatment, the recovery was exceedingly slow, and when the eye eventually became quiet, several unyielding posterior synechiæ and some pigment deposits in the pupillary space were the end results.

Each succeeding year the patient would return with a recurrence in the same eye, and each fresh attack, which was always attributed to "taking cold," left permanent damages in the ocular structures. During the past year, for the first time, an investigation was made of the dental region, despite

the fact that the patient had been assured by his dentist that his mouth was free from infection.

The examination disclosed several beautifully crowned upper teeth on the side of the affected eye and the information was elicited that no *x*-ray examination had been undertaken either before or after the work had been done. A complete *x*-ray examination in this region revealed large abscesses alongside the roots of the left upper incisor and a right lower molar, both of which had been crowned.

The extraction of these teeth and drainage of the abscesses resulted in a rapid amelioration of the eye symptoms, and in a comparatively short period the eye was normal. A few months subsequent to this experience the patient returned with a sharp attack of iritis in the right eye, which before had never been involved. The source of infection was found in an apical abscess of a right crowned bicuspid, and the inflammatory symptoms likewise rapidly subsided after removal and drainage of this tooth, which had not been included in the first *x*-ray examination and was not regarded with suspicion until the second eye became involved.

This case history has been recited at some length for the purpose of emphasizing two points: First, other sources of sepsis being eliminated, the presence of crowned teeth should always be regarded with grave concern, and these should be thoroughly investigated in searching for focal infections in the dental region; and, second, in looking for septic foci the dental *x*-ray examination should not be limited simply to suspected teeth. A complete investigation is necessary in every case in which dental sepsis is supposed to be the causative factor.

Two other cases in the writer's series developed iritis in which the primary source of infection was located in crowned teeth, but it is unnecessary to burden this communication with further case histories illustrative of this point.

The relationship between blind dental abscesses and accommodative asthenopia is discussed by Fulkerson,* who re-

* Amer. Acad. Ophth., Otol. and Laryn., 1916.

ports a number of interesting cases in which the failure to relieve asthenopic symptoms by the correction of refractions and muscular errors was due to the presence of septic foci in the teeth, and in which the accommodative difficulties disappeared when the dental lesions were cured.

The writer has had a few examples of this character and is convinced that dental sepsis is a factor to be reckoned with in a certain number of cases of accommodative asthenopia which resist the usual methods of treatment. In this connection, Knapp* calls attention to the reflex neuralgias located in and about the eye which cause asthenopic symptoms, and for which frequent changes of glasses give no relief until a visit is made to the dentist and a defective tooth is found to be the cause of the condition.

Not only may dental lesions cause accommodative asthenopia and reflex neuralgic pains, but actual loss of the accommodation may occur from focal infections in the teeth. The report of one such case may prove to be interesting:

Mrs. E. J. S., aged thirty-eight years, developed a sudden dimness of vision in the left eye three weeks before her visit to the writer, which showed no signs of clearing. Her general history was not of sufficient importance to quote, except for the fact that she had recently been under the care of a dentist, who had a few days before filled the last tooth which needed attention. This tooth was still somewhat painful and tender and she was ever conscious of its presence.

The dental surgeon, who did not practise roentgenoscopy, had assured her, however, that it was in good condition. Examination showed a normal fundus, clear media, and normal pupillary reaction. Perimetric record disclosed no contraction of fields and the absence of scotomata. Subjective tests: V. L. E. = 20/30, corrected by sph. +0.75. Unable to read Jaeger type but sees clearly Jaeger No. 1 when sph. +3.00 is added. Diagnosis: Paralysis ciliary muscle. The usual physical tests having yielded negative results the importance of eliminating dental sepsis as a possible etiologic

* Med. Ophth., p. 391.

factor was impressed upon the patient, and she consented to undergo an *x*-ray examination. The picture showed an incompletely filled root-canal and a large peridental abscess involving the tooth of which she had complained.

With the hope of saving the tooth, the abscess was opened and drained. While this procedure was followed almost immediately by marked improvement in the eye condition, the accommodation was not completely restored until the tooth was extracted. It has remained normal since.

In this connection it is interesting to note that Theobald,* many years ago recorded two cases of monocular paralysis of accommodation, attended by mydriasis, which were clearly due to reflex dental irritation. In the writer's case, while the loss of accommodation was complete, there was no co-existing paralysis of the sphincter muscle of the iris.

The association between dental lesions and certain diseases of the cornea has been referred to by a number of observers. The writer recalls that during his association with Theobald twenty years ago, this observer not infrequently referred to the influence of dental irritations in those types of keratitis originating from lesions in the ophthalmic division of the fifth nerve or its ganglion. In a discussion of neuropathic keratitis in his text-book, issued in 1906, this author says: "The possibility that an intractable keratitis of the type under consideration may be due to reflex dental irritation should not be lost sight of. Such cases, doubtless, are not common; but that they do occur I am convinced. 'Dead' teeth always, it would seem, on the side of the eye affected and usually in the upper jaw, are more apt to produce such consequences. If, therefore, such teeth, or others badly carious, are found to be present, and especially if they are painful and sensitive to pressure, they should be extracted without unnecessary delay."

More recent studies in the association of dental caries with

* "Prevalent Disease of the Eye," p. 223.

trophic disturbances in the cornea have been undertaken by Blum,* who records seven interesting cases in which the removal of the dental factor resulted in either a marked amelioration of the symptoms or a cure. This author believes that the corneal processes associated with dental irritation are dependent upon trophic changes, and asserts that trophic ulcer of the cornea is not a rare condition, but occurs much more frequently than is supposed.

In the series of cases tabulated by the writer, only one opportunity was presented to study the effects of dental sepsis in neuropathic keratitis. In this case, while the trophic disturbances could clearly be attributed to the presence of seven suppurating roots, it failed to yield to treatment after removal of the offending teeth. The patient, however, was a man seventy-six years of age with a low degree of resistance, and to these factors may be attributed, in a great measure, the failure of the eye to respond after eliminating the source of its infection. It is possible, too, that the dental infection in this case may have been associated with some other infection, which was overlooked, though a careful search was made for other factors.

Reference has already been made to the influence of dental sepsis in the non-suppurative types of choroiditis, but in the purulent form the writer has not been able to find in the literature any mention of its being a possible factor.

Boyle† records a case of metastatic choroiditis in a young mother who had puerperal fever, in which the eye was in the active stage of panophthalmitis two days after the birth of her child, and other writers have now and then called attention to the development of suppurative choroiditis in some of the acute infectious diseases.

The writer's case occurred in a young girl, whom he had under observation for a period of nine years for the treatment

* Oph. Record, vol. xxv, p. 389.

† Jour. Ophth., Otol. and Laryngol., Oct., 1916, p. 845.

of a mild type of choroiditis, of obscure origin, in the right eye. When first seen, at the age of twelve years, she had shown no evidences of syphilitic or tubercular infection, and except for the eye condition, was apparently healthy in all respects.

The influence of dental sepsis in the development of the choroidal lesion had not been considered, and no investigation had, therefore, been made of the teeth. Two years later her glasses were accidentally broken, a fragment of glass cut the right cornea and was followed by a small prolapse of the iris. No splinter of glass had entered the eye. There were no signs of infection, and after excision of the small knuckle of iris the wound healed by first intention.

After seven years of comparative inactivity on the part of the choroidal lesion, and during which period the vision had steadily improved, the patient returned with a well-developed iridocyclitis. There were no evidences of involvement of either the conjunctiva or the old scar in the cornea. A thorough search was at once made for focal infections, including a careful investigation of the dental region. Twelve septic roots were discovered on the side of the affected eye, from most of which, by compression of the gums, a free discharge of pus could be obtained.

The first effect from extraction of these roots and drainage of their bony cavities was most remarkable. There was an appreciable response of the pupil to the action of atropin, beginning signs of the absorption of the inflammatory exudates, and lessening of the ciliary congestion. For two days the eye made steady improvement and it was hoped the infection had been checked. On the third day, however, it became apparent that it was the seat of a deeper infection, and this was rapidly followed by the active stage of panophthalmitis, which finally necessitated its removal.

The exceedingly rapid development of suppurative choroiditis in this case could only be attributed to the very virulent suppurative process in the dental region.

Optic neuritis as a possible sequel of dental disease, particularly when it is dependent on an orbital periostitis or cellulitis following infection of the antrum, has been noted by

several writers. In view, however, of the fact that little mention is made of a development of a papillitis from dental sepsis in the absence of sinus involvement, it would seem well to record the case history of one of the writer's patients:

A. A. B., aged fifty-five years, a farmer, gave a history of occasional attacks for many years of what he thought was malaria, and had noticed a marked impairment of sight in the left eye one week before his visit. The condition grew worse until the vision was reduced to light perception. There was a slight exophthalmos but no limitation of movement and no signs of swelling in the conjunctival or orbital tissues. The pupil was normal in size and in its reaction to light.

Ophthalmoscopic examination revealed a well-marked papillitis, the disc being elevated about three diopters and its margins entirely obliterated. The retinal arteries were much contracted and the veins dilated. One small retinal hemorrhage was seen near the disc, but otherwise the fundus was normal. Transillumination showed normal sinuses. The systolic blood-pressure was 150 and urinary analysis negative.

The patient, whose home was in a distant county, was requested to return, enter the hospital, and undergo the usual physical examination and diagnostic tests. Upon his return, and before entering the hospital, his mouth for the first time was examined. A few carious teeth and a large number of old roots, from which pus was exuding, was evidence sufficient that the patient at least needed the services of a dentist, even if the septic state of his mouth was not the causative factor of the eye condition.

He was immediately referred to a dentist, who discovered several well-defined abscesses, and the rest of the teeth absolutely destroyed from the effects of pyorrhea. Twenty-one teeth were extracted. The following day the patient reported that he was feeling better than he had for years and the vision had increased to 3/200. From this time on the gums and the bony cavities received daily treatments, and marked improvement in the eye condition could be noted each succeeding day.

At the end of fifteen days vision was normal, the nerve outline quite distinct, no appreciable swelling of the disc, and

the retinal vessels of normal caliber. There was no contraction of the visual fields and an entire absence of scotomata. Other than those recorded, no further investigations were made in the case.

The striking features were the rapid improvement in vision, the absence of sinus involvement, and the prompt disappearance of the fundus lesions as soon as the oral septic condition was eliminated. The exophthalmos, which likewise quickly subsided, was no doubt the result of an orbital periostitis or cellulitis of a comparatively mild type, as the usual symptoms which accompany these inflammatory processes were not in evidence.

Conclusions.—Ocular lesions dependent on dental sepsis are not rare, and other factors having been eliminated a thorough search should always be made in the dental region for focal infections.

While extensive pyorrhœa alveolaris is frequently responsible for ocular disturbances, blind dental abscesses have proved to be the source of the infections in many instances in which the symptoms were first attributed to the presence of pyorrhea.

Crowned teeth are frequent offenders in the production of inflammatory states of the eye and should always be regarded with suspicion.

In every investigation for focal infections in the dental region, the *x*-ray examination should include all teeth. A negative roentgenogram of simply suspected teeth is not sufficient to exclude dental sepsis.

While the uveal tract is the most frequently involved ocular structure as a result of lesions in the teeth, the fact should not be lost sight of that every tissue of the eye can be affected from oral septic conditions.

In the present state of our knowledge vaccine therapy in this class of cases has proved to be disappointing, and further investigations will be needed to demonstrate its value.

CAVERNOUS SINUS THROMBOSIS, WITH NOTES OF FIVE CASES.*

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Thrombosis of the cavernous sinus is the rarest of large sinus thromboses, less than three hundred cases having been published since its recognition by Herman Knapp[1] in 1868. MacEwan,[2] whose description of the condition is one of the best, reports but four cases which he has himself seen. Occurring so seldom in the practice of any one man, early decisive recognition may easily fail. Since, however, the only possibility of lessening the extremely high mortality of the disease lies in prompt surgical interference, such early decisive diagnosis becomes necessary. A brief review of the salient facts known about cavernous sinus thrombosis is therefore presented, based upon the literature, which for so rare a condition is quite voluminous, upon five cases from the practice of the writer which are here first reported, and upon ten cases published since Langworthy's study in 1915.

Notes of five new cases:

Case 1.—S. S., a Polish male, aged twenty-five years, was admitted to the Bridgeport Hospital April 16, 1915. Previous history negative. Present history: Seven days ago swelling appeared on the bridge of the nose, extending to both eyelids, especially the left; no obvious infection; no history of trauma. Slight nasal discharge for past three days. Complains of chills and headache only. Temperature on admission at 4 P. M., 102.8°. Physical examination shows a well-nourished man with marked exophthalmos of the right eye and complete immobility of the globe. Pupil dilated and

* Candidate's thesis for membership accepted by the Committee on Theses.

insensible to light. Some swelling of the lids and chemosis. Left eye normal. Pain and tenderness over the entire face and slight stiffness of the neck. Physical examination otherwise negative.

April 17th: Temperature, A. M., 101°; P. M., 105°, restless, complaining of violent headache. Rhinologist reports "nothing in nasal passages." Slight swelling of left upper lid was noted at 5 P. M.

April 18th: Temperature, A. M., 103.4°; P. M., 105°. Marked exophthalmos and fixation of left globe all day. Symptoms in right eye receding. Spinal fluid reported by Dr. Peters: "Pressure slightly increased; turbid; 500–600 cells per c.mm., mostly polymorphs; globulin ++; Fehling present; no organisms." Blood culture shows staphylococcus aureus bacteremia. The patient was semi-conscious in the morning, unconscious in the afternoon. 8 P. M. temperature 106°. At 8.55 he died.

April 19th: Autopsy of head only permitted. Report by pathologist, Dr. Peters: "There is considerable chemosis of tarsal conjunctiva, especially of left eye, but no exophthalmos. On removal of brain there is found a thin, purulent exudate over base. In the cavernous sinus of the right side is found a somewhat purulent bloody substance; no firmly attached thrombus. Ethmoid cells and frontal sinuses normal; mastoids normal. Smears from base of brain and from sinus show numerous Gram-positive cocci, mostly in pairs."

CASE 2.—Mrs. H. G., an American woman, aged thirty-five years, was seen in consultation with Drs. Cogswell and Roller at 1 A. M. August 15, 1915. Previous history negative, except for chronic right ethmoid infection. Present history: Chills and high fever for several days, with severe headache and meningeal symptoms; swelling of right eye thirty-six hours ago and of left eye beginning eight hours ago. Delirious for the past six hours. Physical examination: Patient unconscious but very restless. Marked exophthalmos of both eyes with swelling of the lids, intense chemosis of the conjunctiva and complete immobility of both globes. Pupils dilated and insensible to light. Fundi show considerable congestion and dilatation of veins. Temperature 106°. One hour later she died. No autopsy permitted.

CASE 3.—W. H. L., an American male, aged thirty-five years, was seen in consultation with Dr. D. C. Patterson on October 11, 1915, because of a conjunctivitis accompanying a facial erysipelas which apparently started from a fissure in the right nostril. The conjunctivitis cleared and, owing to absence from the city, the man was not seen again. Upon returning, his physician reported that the erysipelas ran a typical course and was very much improved; but on October 27th he became much worse, the right eye was proptosed, the pupil dilated, and there were marked chemosis and lid swelling. Three days later exophthalmos of the left eye appeared and there were symptoms of meningitis. On October 31st he died.

CASE 4.—Miss A. W., an American woman, aged sixty years, was seen in consultation with Dr. Cogswell October 4, 1916. Previous history negative except for an abscess at the root of the left eye-tooth, which had been under treatment for the past four weeks. Present history: Two days ago there was a slight swelling of the left upper lid which slowly increased; aside from this she seemed well until this morning, when her sister thought she was a little flighty in conversation at breakfast and insisted upon putting her to bed and sending for Dr. Cogswell, who came at noon and found her semiconscious, restless, with a high temperature, and with some exophthalmos of both eyes. At 3 P. M., when I first saw her, there were chemosis, marked exophthalmos, and complete fixation of both globes, pupils dilated and fixed, and retinal veins greatly dilated. She had a high temperature, was unconscious and could not be aroused. At 9 P. M. she died. No autopsy permitted.

CASE 5.—T. M., a German, aged thirty-eight years, seen in consultation with Dr. Roller December 4, 1916. Previous history not obtained. Present history: Sickness began with a boil on the left side of the nose seven days ago. This was incised and dressed dry. Two days ago he had a chill and high fever and meningeal symptoms appeared. This morning exophthalmos of the left eye was noticed, and he became irrational. This afternoon the right eye also was involved. When I first saw him, at 9 P. M., there were moderate exophthalmos of both eyes with complete loss of motility, dilated

pupils, fundi normal except for congestion; some doughy swelling below and outside the left eye, apparently not connected with the boil on the nose, which was healing; patient unconscious and quiet, with deep stertorous breathing. Four hours later he died. No autopsy.

Notes of Ten Cases Reported Since 1915 or Not Mentioned in Previous Studies.—Wacker's case[18] followed a furuncle inside the nose. Exophthalmos on the third day, meningitis the fifth, death on the seventh day. Autopsy showed staphylococcic thrombophlebitis of both cavernous sinuses and metastatic abscesses in the lungs.

Henning's case[19] was also from a facial infection, as was another case reported in the discussion.

Dayton [20] reports two cases. One from a purulent infection in the nose with death eleven days after eye symptoms appeared; the other in a girl of fifteen who had typhoid fever and a probable lighting up of a chronic nasal sinus disease; death in seven days. No autopsies.

Payr[21] diagnosed a sterile thrombus in the cavernous sinus following an infective thrombophlebitis in the upper arm of the opposite side. There was no exophthalmos, but all the nerves in the sinus were involved.

Davis' case[22] followed tonsillitis, with death in ten days. Exophthalmos of one eye only. Spinal fluid normal. He has seen two other such cases after tonsillitis, one recovering with loss of sight, the other dying on the fifth day. Also a case with double exophthalmos following mastoid disease and operation, with death on the fifth day. In the discussion O'Malley reported a case three weeks after tonsil and adenoid removal; autopsy showed osteomyelitis of the sphenoid, probably the primary focus. St. Clair Thompson had seen three cases, two from the sphenoid and one from the ear. J. Horne had seen one sphenoidal case. F. Powell had seen one case secondary to intracranial suppuration.

Sitzen[23] reports two cases. The first followed a boil on the nose, with death ten days after its appearance and three days after exophthalmos had involved the second eye. Autopsy showed thrombosis of the cavernous and superior petrosal sinuses, and meningitis from staphylococcus infection. The second case followed a facial erysipelas starting in a nasal furuncle. There were exophthalmos of both eyes and

pronounced meningeal symptoms. Autopsy showed cavernous sinus thrombosis and meningitis. Knoch reported a third case, also from a boil on the nose, with death on the third day. No autopsy. These three cases occurred in Java.

Surgical Anatomy.—The cavernous sinus, the most important venous channel at the base of the skull, lies between layers of the dura beneath the temporosphenoidal lobe. It is roughly quadrilateral in shape, larger behind than in front, 2 cm. or more in length and about 1 cm. in diameter. Evidences of its origin as a venous plexus in the dura remain in the interlacing bands of lining endothelium, which divide it into several irregular compartments. It extends along the side of the sella turcica, separated by a very thin bony wall from the sphenoidal sinus of the nose, from the sphenoidal fissure at the apex of the orbit, where it receives the ophthalmic vein, to the apex of the petrous portion of the temporal bone, where it opens into the petrosal sinuses—the superior to the lateral sinus and the inferior to the jugular vein. The cavernous sinuses of the two sides are connected by the circular sinus, two transverse vessels, one in front of and one behind the pituitary body. On the internal wall of each sinus lie the internal carotid artery and the sixth nerve; on its outer wall from above downward the third, fourth, and the ophthalmic division of the fifth nerves. Each cavernous sinus has emissary veins through the foramina ovale, lacerum medium, and Vesalii to the pterygoid and pharyngeal plexuses, and a plexus of veins around the artery in the carotid canal to the internal jugular vein. It also receives veins from the frontal lobes of the brain and from the middle cerebral vein.

The cavernous sinus is thus in direct venous connection through the lateral sinus with the veins of the ear, through the pterygoid plexus with the veins of the mouth and throat, and through the ophthalmic vein with the veins of the orbit, nose, and face. The facial vein, on account of its exceptional

patency and lack of valves, favors septic absorption and thrombus formation, and the extension of thrombi therefrom to the cavernous sinus is anatomically easy, either *via* the angular and ophthalmic veins, or *via* its tributary from the pterygoid plexus. The orbital veins are also valveless. The inferior ophthalmic, which has a branch through the sphenomaxillary fissure to the pterygoid plexus, joins the superior ophthalmic, which communicates with the angular vein at the root of the nose and receives blood from the ethmoidal veins and the venæ vorticosæ, and empties into the cavernous sinus together with the central vein of the retina.

Etiology.—According to MacEwan, purulent otitis is the commonest cause of cavernous sinus thrombosis. Next to this are infective lesions of the orbit and face, *i. e.*, of the area drained by the ophthalmic vein and its branches. Of these, orbital cellulitis, boils, and infected wounds on the face, and facial erysipelas are the most common. Other causes are purulent affections of the nose and accessory sinuses, especially the sphenoid and the ethmoids, pharyngeal and tonsillar infection, and abscesses at the roots of the teeth. Thrombosis may occur from trauma in the immediate vicinity of the cavernous sinus, as in head injuries. A non-infective thrombosis of the cavernous sinus has been described by Knapp.

The following table shows the original source of infection in 140 cases:

	Ear	Orbit and Face	Mouth and Throat	Nose	Other Causes
Dwight-Germain [4]	43	40	14	9	0
Jackson [5]	13	6	2	0	4
Cases here reported	0	3	2	4	0
Approximate per cent.	40%	35%	13%	9%	3%

Infective meningitis from caries of the body of the sphenoid may extend by contiguity, according to MacEwan,[2] directly

to the cavernous sinus, as may intracranial suppurative processes. Ordinarily, however, there is set up in the veins draining the infective source a thrombophlebitis, from which the thrombic process extends through venous channels to the cavernous sinus. Its usual routes are from the sigmoid through the petrosals from behind, through the ophthalmic veins in front, or from below by way of the pterygoid plexus. The infecting organism may be any one of the pus-producing group, of which perhaps the staphylococci and streptococci are most frequently found.

Symptoms.—The general symptoms are those of any infective sinus thrombosis—headache, chills, fever with a steep-peaked chart, quick soft pulse, and euphoria. Occasionally there is vomiting. Leukocytosis is present; blood infection may or may not be found. Spinal fluid changes, if present, are those of a beginning basilar meningitis, which is not uncommon. Pulmonary complications from septic emboli in the lungs are frequent. The later symptoms are those of a rapid, profound sepsis, delirium, then coma preceding death.

The local symptoms are largely eye symptoms, and occur in two groups: (1) Those due to intra-orbital venous obstruction, and (2) paralysis of the nerves within the sinus, due to pressure and to phlebitis of the sinus wall.

Of the first group, the most important as well as one of the earliest symptoms is exophthalmos. This, according to most observers, is rarely absent; Knapp[6] states that it occurs in 72 per cent. of the cases. It appears first in the eye upon the same side as the originating infection, rarely on the opposite side, and increases as the infection advances. The eyeball is pushed forward by the venous engorgement and edema at the back of the orbit, which also cause swelling and edema of the lids, extending over on to the root of the nose, chemosis of the conjunctiva, and engorgement of the conjunctival veins. This may be well marked within a few hours. If the

thrombic process involves the pterygoid plexus, there may be edema of the pharynx and tonsil, but this is easily overlooked unless the latter was the source of the infection. There may also be edema of the face from obstruction of the facial vein and of the tissues of the temple and about the ear. All these obstructive symptoms vary in amount in different cases, being particularly marked where there is ophthalmic phlebitis; they may entirely disappear with the development of the collateral circulation.

Shortly before death the same symptoms usually appear in the other eye, often with a recession of symptoms in the first eye. The involvement of the second eye indicates the extension of the thrombus through the circular sinus to the cavernous sinus of the other side, and makes the diagnosis practically certain. MacEwan[2] says that involvement of the second eye occurs in more than half the cases. One hundred and thirty-four autopsies collected by Dwight-Germain[4] showed only 31 in which the thrombosis was limited to one cavernous sinus.

The second group of local symptoms comprises the various paralyses of the nerves within the cavernous sinus. The third nerve is most often implicated, ptosis being one of the most constant symptoms, though often not available for diagnosis on account of the great edema of the upper lid. Strabismus varies, depending upon the priority of involvement of the third, fourth, and sixth nerves, and soon gives way to complete ophthalmoplegia.

The various paralyses may occur early, and are best observed in the cases of slow development, particularly those in which the infection comes from behind. In the more acute cases, and in those in which the infection comes *via* the ophthalmic vein, the displacement and fixation of the eyeball from intra-orbital pressure often occur before any distinct paralyses can be recognized. The pupil, small at first, is widely dilated, sluggish, then immovable. The cornea be-

comes hazy and anesthetic and may break down from exposure. Increased lacrimation, according to Weeks,[7] is soon followed by comparative dryness of the conjunctiva. Neuralgic pain in the first division of the fifth nerve may occur, but is much less common than headache.

Vision fails or is lost according to the pressure on or stretching of the optic nerve. Its measurement, however, is usually impracticable on account of the general condition of the patient. In four cases which recovered, quoted by Jackson,[5] only one had good vision, and in Johnson's[9] and Davis'[22] cases vision was lost.

The ophthalmoscopic symptoms are not ordinarily important and may be lacking, probably because of the free anastomosis of the ophthalmic veins with the facial and with the pterygoid plexus. If the fundus can be seen, fullness and tortuosity of the retinal veins are usually found, with perhaps slight blurring of the margin of the disc; neuroretinitis and retinal hemorrhages may occur. These are symptoms of obstruction of the ophthalmic and retinal veins rather than of cavernous sinus thrombosis. The course of the disease seems ordinarily too rapid to allow the development of a well-marked neuritis.

Diagnosis.—Septic thrombosis of the cavernous sinus will be thought of whenever an exophthalmos shows also septic and cerebral symptoms, or any case of probable cranial sinus thrombosis develops orbital symptoms. The combination of lid swelling, conjunctival chemosis, exophthalmos, and paralysis of a nerve within the sinus on the same side as an infective lesion anywhere about the head is quite characteristic, and the diagnosis is made certain by the similar involvement of the other eye.

When the infection comes from behind or below, the process extends from the cavernous sinus to the orbit, and the general symptoms of sinus infection precede the local or orbital ones. Here the first appearance of the characteristic

local symptoms makes the diagnosis easy, and an earlier diagnosis is possible than in the cases infected from the front. In the cases where the infection is by way of the ophthalmic vein,—as, for example, where the sinus involvement originates from a previous orbital cellulitis,—orbital symptoms precede the sinus infection. Here the involvement of the sinus is marked by the deepening of the symptoms of the cellulitis, and the addition of those of cavernous sinus infection. In these cases it is more difficult to be sure of the diagnosis until the second eye begins to be involved.

The precise order in which the symptoms appear may thus depend upon the source and path of the infection. Familiarity with the possibilities of infection in this region and a clear mental picture of the pathology of sinus thrombosis are of more service than statistics of symptoms in making an early diagnosis.

Differential Diagnosis.—Septic cavernous sinus thrombosis is to be differentiated from other pathologic processes within the cavernous sinus. These, according to Langworthy,[3] are injuries from gunshot wounds, from fractures at the base of the skull, and from operations on the Gasserian ganglion, the hypophysis, and the sphenoidal sinus; arteriovenous aneurysm; malignant or congenital new-growths involving the sinus; marasmic or aseptic thrombosis; and perhaps intermittent exophthalmos. In all these the history, the rate of development, and the other symptoms should afford basis for diagnosis.

Infective sinus thrombosis is to be further differentiated from pathologic processes within the orbit. Of these, orbital cellulitis and orbital thrombophlebitis limited to the orbit (the latter, according to Weeks,[7] occurring practically only in erysipelas) offer the most difficulty. These have, however, no cerebral symptoms unless the process extends to become sinus thrombosis, meningitis, or cerebral abscess.

Exophthalmos and other symptoms which might possibly

cause confusion are also present in: (1) Tenonitis, which is a much milder condition, often suggesting rheumatic infection, and associated with trauma or with infectious disease rather than with an infective focus. (2) Periostitis, with thickening and tenderness of the orbital periosteum and with less interference with eye movements. (3) Panophthalmitis, with the infection mainly inside the globe. (4) Dacryo-adenitis, a rare affection. (5) Infection of the nasal accessory sinuses adjacent to the orbit. (6) Exophthalmic goiter. (7) Tumors and cysts of the orbit, of the optic nerve, and of the lacrimal gland.

Course.—In infective cases the obstructive symptoms, exophthalmos, lid edema, and chemosis, develop early and gradually, and ordinarily reach their maximum in two to five days, rarely ten or more, when they begin to subside. The development of the process in the second eye is a late symptom and is much more rapid, being often a matter of hours. The nerve symptoms come early and are soon masked by the obstructive symptoms. The sepsis grows more profound, and embolic processes, especially in the lungs, are frequent. Meningeal symptoms and those of increased intracranial pressure often develop, and death regularly follows from sepsis in from six to forty-eight hours after the involvement of the second eye.

Autopsy Findings.—Autopsy findings by various observers agree only in that the cavernous sinus contains a thrombus, often partially disintegrated, or a purulent fluid; that usually both sinuses are involved; and that there is frequently a basilar leptomeningitis. Other findings depend upon the source, route, and nature of the infection.

Prognosis.—The process is always fatal if the thrombus is infected and is not drained. If the symptoms arise simply from an obstructing sterile clot by extension from the sigmoid sinus, and this clot does not become infected because of early operative interference on the sigmoid, recovery may

be possible. This has actually occurred in three cases quoted by Dwight-Germain,[4] in one by Adair-Dighton,[8] and in one by Johnson.[9] Bircher[10] went through the petrous portion of the temporal bone and drained pus from the posterior end of the cavernous sinus successfully. Langworthy[3] quotes three recoveries from drainage of the infecting source only; in these it seems fair to assume that the thrombus in the cavernous sinus was not infected.

In some such way must the few recoveries, where septic thrombosis of the cavernous sinus has been diagnosed, be explained. The total of recoveries in nearly 300 cases reported in the literature* amount to 23—about 7 per cent. Eliminating those recoveries which may be explained as above, and considering the great number of observers, the fragmentary character of many of the reports, and the possibilities of inaccuracies and of errors in diagnosis, we are forced to the conclusion that thrombosis of the cavernous sinus is practically always fatal if the thrombus is infected and is not drained.

Treatment.—Except for drainage of the infective source, and drainage of the lateral sinus in ear cases, the treatment of cavernous sinus thrombosis is so far practically nil. The only hope of effectual treatment lies in the analogy of this condition to thrombosis of the lateral sinus, where early diagnosis and prompt, bold, and thorough surgical intervention saves many lives. Theoretically, if the cavernous sinus were attacked exactly as the otologist attacks the lateral sinus, by free incision, removal of the clot or its septic part, and drainage, some cases should recover, especially those in which diagnosis and operation are made early. The difficulties in such a procedure are obvious. Lateral sinus infection is common and its operative technic has been well

* The exact number of cases reported is uncertain, as collections by different authors contain a few of the same cases, and sufficient data are not always given to surely identify all of these.

developed, while cavernous sinus thrombosis is too rare to allow any surgeon to see more than a few cases. By the time the diagnosis is usually made and the question of operation arises, the sepsis has progressed so far as to make the chances of recovery slight. The cavernous sinus, too, is surgically one of the least accessible structures in the body, and few operators care to explore it. The feasibility of its drainage has, however, been demonstrated in operations by Hartley,[12] Dwight-Germain,[4] Ballance,[13, 27] Ballance and Hobhouse,[13] and Voss,[14] all of whom approached the sinus by the Hartley-Krause Gasserian ganglion route, through the side of the skull; by Mosher[15] and Adair-Dighton,[24] going through the orbit; and by Bircher,[10] in an ear case, going through the petrous portion of the temporal bone to the posterior end of the sinus. The last case was the only one to recover. This, however, is not a fair measure of the success of the operation. In some cases the operation came too late to relieve the sepsis; in some the sinus itself was missed. In Hartley's case a sarcoma was found in the sinus, and this was the cause of the patient's death two months later. In Dwight-Germain's case, aged forty, incision into one sinus completely relieved the interference with the circulation in both sinuses. The drainage of one sinus in Adair-Dighton's case, aged six, did not drain the other, but the original focus in the nose was overlooked.

Other routes to the sinus seem possible. Luc proposes an extension of his operation for exposure of the maxillary antrum. Beck has successfully operated hypophyseal tumors by way of the antrum, following Jansen's method. Langworthy,[3] as a result of cadaver study, proposes a nasal route through the roof of the sphenoidal sinus, a route which Hirsch and others have successfully followed in operations upon the pituitary body in acromegaly.

Of the possible routes by which the cavernous sinus may be surgically reached, that from in front is in the field of the

ophthalmologist, those from below in that of the rhinologist; the brain surgeon has developed entrance through the side of the skull, and one otologist has reached the sinus from behind. The weight of evidence so far favors the Gasserian ganglion route, though the route through the sphenoidal sinus may be more attractive to those who have gained experience in this region by operating upon the pituitary body. It may prove that the choice of routes will be that with which the operator is most familiar, or perhaps that by which the infection has entered. The observations of military surgeons in injuries of the cavernous sinus may throw light upon the question. Meanwhile we do not know how many of these theoretic considerations are practical.

Operative interference has, so far, few advocates and fewer practitioners, though its feasibility is admitted and it seems to be the only rational treatment when it has been better worked out. With many surgeons of experience the objections to operation outweigh its advantages, though this fact may be discounted by the natural conservatism of the profession toward all unproved measures. Opinion is not unanimous. Many agree with de Schweinitz,[26] who thinks that "operation would seem to be proper," and with Frazier, who is quoted by Pooley[25] as saying that "direct attack on the cavernous sinus should be made in the hope of saving more lives."

Langworthy[3] makes the point that, until we have a better understanding of the very early signs of the disease, we cannot expect to improve in either diagnosis or surgical treatment. This should be gained by a more general alertness and more careful observation in that class of infections from which thrombosis of the cavernous sinus may develop.

Conclusions.—A study of septic cavernous sinus thrombosis indicates:

1. The hopelessness of any but surgical measures—first, to the source of infection, second, to the sinus itself.

2. Septic cavernous sinus thrombosis is an operable condition. The sinus should be opened and drained if a definite diagnosis can be made before the patient becomes too septic.

3. An early definite diagnosis can be made in at least some cases if the original observer is familiar with the condition and is sufficiently alert. Discussion of the subject may therefore be of value, as many cases doubtless go unrecognized. More careful study of the earlier symptoms is needed, and the possibility of sinus thrombosis developing from apparently trivial infections about the face and head kept in mind.

4. A larger number of cases operated upon is necessary before more definite conclusions can be drawn. In view of the hopelessness of non-operative measures, and the possibility of saving an occasional case by early and thorough drainage of the sinus, the operation should be undertaken whenever there is any reasonable possibility of success.

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HYPERPLASTIC EXUDATIVE RETINITIS—NON-HEMORRHAGIC.*

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True hyperplastic, exudative, non-hemorrhagic phenomena in and apparently adjacent to the retina are rare. The appearances which they present to ophthalmoscopic examination are frequently similar to, and perhaps often indistinguishable from, the lesions resulting from previous hemorrhagic extravasations. In fact, the ultimate result, subjectively and objectively, of organized hemorrhagic and non-hemorrhagic exudates might conceivably be pathologically identical, as in the lesions of retinitis proliferans, which may have their origin in either form of exudation.

This latter form of retinitis is, of course, described in all modern text-books, but the rarity of its occurrence may be realized by the statement of Weeks that he had seen but 2 cases in 24,000 private patients, and of Schöbl, quoted by Weeks, who observed 5 cases out of 179,057 patients. The common acceptance of its etiology is hemorrhagic extravasation from the retina into the vitreous. It has been pointed out, however, that it may occur consequently upon the ejection of a fibrinous exudate or coagulum, with or without the presence of blood-corpuscles (Weeks).

Leaving out of consideration, for the present, cases of retinitis circinata, which will be referred to later, the writer has observed at least four cases showing lesions, in six eyes, of what appeared to be true organized retinal exudates, non-hemorrhagic, three of them being confined within or subja-

* Candidate's thesis for membership accepted by the Committee on Theses.

cent to the retinal layers, and one combining this type with the currently described type of retinitis proliferans extending into the vitreous body. In none of these cases was there any evidence of previous or present hemorrhage at the site of the lesions, and in all except one the development of the processes was watched from week to week and month to month under ophthalmoscopic observation.

In view of these observations, which are detailed below, the writer suggests that proliferating or granulating or chronic inflammatory (neoplastic) tissue may occur within as well as without (with an origin within) the retinal layers; that it may occur without preceding hemorrhagic phenomena; and that whether within or without the retinal layers these lesions may properly be classified as hyperplastic exudative (non-hemorrhagic) retinitis.

From all that is known of retinitis circinata, it would seem justifiable to include this type in the above classification, the difference in the ophthalmoscopic picture being due merely to the different arrangement of the lesions.

The identification of hyperplastic exudative retinitis with the conditions described by Coats in 1909 as a "disease of the retina, hitherto insufficiently differentiated, which is characterized by the presence in the fundus of large masses of white or yellow exudation; usually there are also groups of cholesterin crystals and hemorrhage" (massive exudation of the retina—Coats), seems more than possible. There seem to be, however, some interesting points of difference. For instance, Coats states that "in the only case of this disease which has so far been examined in a recent stage, the white mass in the fundus was found to be due to a disintegrating hemorrhage." In three of the subjoined cases the growth and development, and in one case the retrogression, of the lesions were observed by the writer over a considerable period, and at no time could hemorrhage be suspected of having a part in the origin or progress of the lesions.

Coats remarks that "in an allied form of the disease, or in a variety of the same form, strange types of vascular disease are found,—fusiform and globular expansions, kinks, loops, glomeruli, etc.,—as well as the more common types, such as constriction, white ensheathing, etc." Interestingly enough, this "allied form" occurred in the left eye of case 1, described below, the right eye having been enucleated after destructive blindness had ensued on a continued development of the hyperplasia and excessive glaucomatous tension.

In at least two of these cases, 2 and 4, a neogenetic vascularity was observed as a part of the hyperplastic processes. This vascularity has an apparent tendency to disappear from ophthalmoscopic view after a longer or shorter lapse of time, but whether from actual resorption or from becoming obscured by cellular infiltration of leukocytes or the development of fatty degeneration, it seems difficult to determine, though the writer inclines to the two latter hypotheses. The evidence leading to this conclusion was quite marked in the case of the enucleated right eye of case 1. Here the chief lesion was about the entrance of the optic nerve. In enucleation the nerve was severed three or four millimeters behind the bulb; and surrounding the nerve, beneath the sheath, and giving it a somewhat lobulated appearance more than twice its normal size, was a mass of what looked macroscopically like chronic inflammatory or granulation tissue, with finely spun vascular elements—certainly well-organized hyperplastic tissue, and certainly a part of the process ophthalmoscopically observed; and from indications at the time, proved by subsequent events, evidently not malignant. There is no doubt that this process extended further up the nerve than the point of severance. In case 2 a well-marked, fine vascularity appeared in the exudative mass, subsequently disappearing from ophthalmoscopic view.

In none of the writer's cases was there any of the stigmata of lues, inherited or acquired, and repeated examinations

clinically and by laboratory methods of cases 1, 2, and 4, failed to show positive signs of either syphilis or tuberculosis. In none was there a history of trauma obtainable, except as noted in report of case 2. It is true that in case 1, while the second eye was giving us some concern, Dr. G. E. de Schweinitz, who was the consultant in the case, reported that there was a strong positive reaction to the von Pirquet test. However, this was subsequent to repeated subcutaneous and von Pirquet tests previously made (with negative results) under the writer's direction, and the possibility of anaphylaxis may be properly considered. Dr. de Schweinitz quotes the clinicians' report as follows: "Although the physical signs at the right apex are not sufficient in themselves to make a diagnosis of tuberculosis, and an *x*-ray examination is always a little uncertain, when these are taken together there seems little doubt that a lesion in the upper lobe exists, and the probability is that the lesion is a tuberculous one." Fifteen months have elapsed since this report was made, and no further evidence of tuberculosis has been obtainable.

In all the cases except 3 (who was seen only twice) all possible points of focal infection were satisfactorily eliminated from consideration as etiologic factors except as follows: Case 1 had suspicious faucial tonsils, which were enucleated; and later *x*-ray examination of the various sinuses gave apparently positive evidence of an old right mastoid infection. This mastoid was opened and cleaned out, but there proved to be little evidence of diseased processes present. In case 4 it was discovered that active pus tonsils were present, and these were promptly enucleated. The effect of the removal of these foci in case 1 is rather difficult of estimation, but, on the whole, it may be said that the result appears satisfactory. At any rate the remaining eye is in a more quiescent condition since this interference and vision is normal with correction. In case 4 a sufficient time has not yet elapsed since the removal of the tonsils to warrant a

statement of intraocular effects, though the patient expresses herself as feeling much better generally.

In these cases the circulatory, intestinal, and kidney functions were repeatedly, and the writer believes carefully and thoroughly, investigated with negative results in informative value. Cases 1 and 2 showed undoubted benefit from massive doses of iodid of potash, but this can hardly be taken as indicative of anything definite as to the etiology.

After observing for several months the development of the process in case 1, right eye, the writer had made a diagnosis of probable malignant tumor. Dr. de Schweinitz, however, after a careful study of the case suggested "massive exudation of the retina," or else the rarer type described by von Hippel under the name of "angiomatosis retinæ," but concurred in the advice to enucleate the eye, "inasmuch as in either circumstance the eye is sure to go on to secondary glaucoma and complete blindness, and as a staphyloma is already beginning to form in this eye." The writer is now convinced that de Schweinitz's diagnosis of massive exudation was practically correct.

With these cases, as suggested above, it would seem proper to classify, clinically, those conditions of exudative, non-hemorrhagic retinitis which are typified by the so-called retinitis circinata, retinitis proliferans of non-hemorrhagic origin, and the hyperplastic retinitis of syphilitic and traumatic origin which are not of hemorrhagic formation. The cases of the type which the writer is reporting (like retinitis circinata and retinitis proliferans of non-hemorrhagic origin) must be regarded for the present as idiopathic—much as we may dislike the term; though we must necessarily accept the dictum that may be tersely put in this form: In the presence of exudate there must be a departure from the vascular normal. This, for the present, is as far as we can get etiologically.

The suggested classification would come entirely within

the purview of Fuchs' statement* of the anatomic changes in retinal inflammation, and would serve to clarify and make more readily understood the diagnosis of certain rarely observed and hitherto seldom recognized conditions whose ophthalmoscopic changes and pathology have been veiled in more or less obscurity.

(It is a matter of deep regret to the writer that, owing to a mistake on the part of his pharmacist in furnishing too strong a solution of formaldehyd, the eyeball enucleated in case 1 was rendered unfit for laboratory examination, though Dr. de Schweinitz and his pathologist made every effort to use it.)

ABSTRACT OF CASES.

CASE 1.—June 10, 1912: C. S. C., white, male, aged fifteen years. School boy. Excellent family and environment. Right eye went suddenly blind (in space of two or three days) two months before this date. Constant severe parietal headaches right side. Health otherwise good. Nothing of interest in previous personal or family history, except that his father died several years previously of some obscure stomach trouble (probably malignant). V. R. E. = l. p.; L. E. = 20/30, sph. −0.50 = 20/20. No external evidence of disease. Ophthalmoscopic examination of right eye reveals severe advanced type of neuroretinitis with macular exudations, probably going on to atrophy. Left eye shows beginning papillitis. Tension right eye equals +1 (palpation). Clinical examination shows no evidence of any sinus involvement. After several days' treatment at hospital with pilocarpin sweats and eliminants, the right eye is acutely inflamed (uveitis). Excessive tension and eye painful at times. Urinary, blood, heart, chest, and general systemic examinations negative.

During July subconjunctival injections of 15 minims of 1: 5000 cyanid of mercury with ½ of 1 per cent. acoin were given. At end of month inflammation slowly but steadily improving. On August 5th dismissed from hospital. Right optic disc edematous, pale, and elevated about 3 mm. On

* "Textbook of Ophthalmology," translated by Duane, fifth edition, p. 589.

August 26th suspicion of exophthalmos in right eye. Sclera still much congested. Anterior chamber shallow. Pupil 4 mm. diameter. Nearly entire visible fundus white, hazy, and elevated 3 to 4 mm.

On September 10th large white area with heavy bunches of vascular formations seems to be growing steadily in right fundus, now occupying almost the whole visible fundus and best seen with +13 lens, indicating elevation of over 4 mm. Eye totally blind. At this time I advised enucleation of the eye with a probable diagnosis of malignant growth and also suggested a consultation. Dr. G. E. de Schweinitz was consulted, with the result that he concurred in my advice as to an enucleation, but differed in the diagnosis, as stated above in this article. Previous to this time von Pirquet and subcutaneous tuberculin tests had been made under my direction with negative results.

The eye was enucleated on October 24th. At this time the tension had increased to such a point that in places it was evidently telling on the integrity of the eyeball, and the elevation of the growth in the fundus was 5 mm. (The macroscopic appearance of the bulb and severed nerve is referred to in the previous part of this article. As stated above, an error on the part of the pharmacist in supplying a solution unfortunately ruined the eyeball for microscopic examination, and indeed even for gross examination. This was a serious loss, as opportunities for such examinations in this type of case are undoubtedly rare.) Patient made an uneventful recovery, and has been wearing artificial eye comfortably ever since. V. L. E. = 20/15.

January 25, 1913: The history of the left eye from this date was as follows: V. = 20/20, with cyl. −0.25 axis 45° = 20/15. In May the vision remained the same, but there was slight haziness at the edges of the papilla, and the veins were rather large. In October V. with cyl. −1 axis 180° = 20/20. October, 1916, retinal congestion, whitish, glistening corrugations and roughness in region between papilla and macula, and suspicion of stellate streaks about macula. Excessive formation of tortuous fibrillary blood-vessels in this region. V. with sph. −1 ⊃ cyl. −0.50 axis 180° = 20/20. Heart and kidneys normal. Double tonsillectomy in November. Dr. de Schweinitz again saw the patient, and reported: "The

large, string-like band of white tissue about the disc and the oval hemorrhages made me think, in view of the condition (which had previously obtained) of the other eye, that an early proliferating retinitis is beginning." To this letter I promptly replied that when I had last seen the patient, two months previously, neither of these lesions were present. In January, 1917, Dr. de Schweinitz wrote me suggesting a probable diagnosis of tuberculosis, as referred to in the first part of this article. In February, 1917, I again saw the patient and noted in the fundus "suggestions of proliferation" along the upper sweep of one or two of the temporal vessels. This was somewhat in the nature of hazy sheaths running parallel to the vessels, but the "string-like" bands observed by Dr. de Schweinitz had disappeared. The patient was put on increasing doses of iodid of potash and vichy water, and was allowed no use of eye for near work. In March there were in the extreme upper portion of fundus several patches of what look like spots of choroiditis with some pigmentary deposits and crossed by an evidently adventitious and tortuous vascularity. The suspected proliferative signs along the upward sweep of the vessels still persist.

During March and April the treatment was continued. On April 16th a fresh hemorrhage was found to the nasal side of the choroidal patches, about one-eighth size of disc, which later slightly increased. In October the *x*-rays showed an old affection of the right mastoid. The mastoid was opened in November, with findings referred to in first part of paper.

At the present time, April, 1918, the eye is quiet. Vision, with correction, equals 20/15. The patches in the upper fundus are much smaller. There is some tortuosity of numerous small vessels. The faint parallel streaking persists along one vessel in the upper fundus, and the apparent hemorrhage, which is now not more than 1 by 3 mm. in size (ophthalmoscopic magnification), still remains as a bright red spot. I have come to the conclusion that this is not a hemorrhage, but a small aneurysm. All treatment has been discontinued, but the use of the eye for near work has been restricted.

It would seem that this interesting case was one of true

hyperplastic exudative retinitis of non-hemorrhagic origin in the right eye, evidently not a malignant growth, as subsequent events have proved; and in the left eye one of the "allied forms" of the same disease to which Coats has referred. Evidently the vigorous treatment with iodid of potassium was of decided benefit, for the process has undoubtedly not only been checked, but has undergone at least partial resolution.

CASE 2.—April 25, 1916: S. W. F., aged thirty years. Farm overseer. Three days ago noted sudden diminution of vision of left eye. Has had frequent, but not severe, pain "behind eyes." Had fall on back of head about one year ago, stunning him. V. R. E. = 20/20; L. E. = 20/200. Homatropin and retinoscopy show practically emmetropia. Ophthalmoscope shows slight fluffiness of papilla, left. Faint yellowish reflex over macular area. Tension normal. General health excellent. Positively denies specific history. Does not use tobacco or alcohol. Left pupil slightly larger than right. Does not react as well to mydriatic. Possibly intracranial tumor or ruptured vessel. Ordered iodid of potassium in increasing doses.

May 1st: V. R. E. = 20/20; L. E., fingers, 4 feet, central vision. He seems to have a fair eccentric field. Ophthalmoscope, left, shows a disc of white over macular region, about size of papilla; hazy edges and a few minute vessels, not elevated. I noted a peculiar, faintly yellowish reflex at this point a week ago, evidently a tumor. Wassermann reported negative. General examination, including chest, heart, kidneys, etc., negative.

May 26th: Exudate is irregularly oval and somewhat lobulated. No longer any trace of vessels on this formation. Seen clearest with sph. −2 in ophthalmoscope. Evidently subretinal. Whole fundus slightly hazy. Taking 32 drops of iodid of potassium. Ordered continued increase. The exudation above referred to is about the diameter of the disc horizontally, and a little over half of this measurement in the vertical line. I think undoubtedly one of the rare cases of massive exudation of the retina.

June 2d: Macular patch undoubtedly smaller, being less

than a disc diameter horizontally and less than one-half of this vertically. Considerable dust-like opacities in vitreous.

June 20th: Improving. Exudative mass about one-half size of a month previous, about same general outline. Shades off at periphery as if undergoing absorption. Now taking 80 grains iodid daily.

July 1st: Exudate slowly absorbing. White, glistening area less than one-fourth former size and somewhat triangular shape, shading off over original area into normal retina.

July 15th: Few sparse, punctuate, glistening retinal spots just above central fundus. Central lesion slowly absorbing, with slight tendency to pigmentation. Patient again declares absolutely no tuberculosis has ever existed in any branch of family.

In September improvement continued. V. 20/200, eccentric; iodid stopped. Left macula now appears as having faint, grayish appearance, in size one-half diameter of disc horizontally and one-third disc diameter vertically; ovoid, and at lower center a darkly pigmented spot. In October vitreous clearer; macular patch smaller and less distinct, and more regular ovoid shape, and about one-eighth area of disc in size.

There can be no doubt that this was a case of exudative hyperplastic formation of organized tissue. The unusual opportunity was here presented of observing the clinical development from the time vision was first seriously impaired by what was evidently a retinal or subretinal fibrinous or plastic exudate, to an organized vascular formative period, and on to final partial retrogression, with undoubted permanent functional impairment. At no time was there the slightest trace of hemorrhagic phenomena participating in the origin or growth of the lesions. The patient did not return as instructed, but the reporter is endeavoring to get in touch with him.

CASE 3.—November 30, 1915, Mr. D. C. C., aged thirty-five years, bookkeeper, complains of asthenopia, frequent styes, and chalazia. V. R. E. = 20/20, with cyl. +1.50, axis 90° = 20/20; L. E. = 20/70, with cyl. +1.50, axis 90° = 20/20.

Ophthalmoscopic examination negative, except perhaps rather hazy discs.

On December 7th right fundus somewhat congested and slight "knobby" appearance of papilla. Left fundus shows papilla markedly "knobby" and gray and white mottled, much like a piece of popcorn, with faint suggestion of vascularization. Apparently exudative (?) condition around papilla, and latter is distinctly elevated (3 D.). "Massive exudation of retina." (?) Ordered Crookes' glasses and advised careful watching and further examination. (This patient was of the neurotic type, and became dissatisfied when I told him his trouble might be serious. He did not return after this second visit, although his family physician made every effort to get him to do so. Eighteen months later his physician told me he was complaining of his eyes, but would do nothing for them.)

This case, in the space of seven days, showed a transition from slight papillary haze to a well-marked organized tissue formation without the slightest evidence of hemorrhagic antecedence, nor had his family physician, a very capable man, ever suspected lues.

CASE 4.—September 29, 1917: Mrs. W. M. E., aged fifty-five years, finds vision has recently been failing. No pain or inflammation in eyes. General health good. For some years has been wearing reading and distance glasses, fitted by opticians; the present ones recently fitted. V. R. E. = 10/200, with sph. $-2 = 20/100$; L. E. $= 20/20$, no improvement with any glass.

Ophthalmoscopic examination reveals in the right eye a very edematous retina with a few bright yellow spots (cholesterin ?), one to two millimeters in diameter (ophthalmoscopic magnification), in macular area, and between that and disc. The disc is surrounded by an area of white with grayish clouding (probably edematous infiltration). There is a persistent canal of Cloquet, the anterior end of which is quite opaque. Tn.(palpation). Left eye, edematous retina and disc. Tn. There is no sign, symptom, or history of syphilis, tuberculosis, or trauma, and no indication of hemorrhages.

Family physician reports heart, kidneys, and blood-pressure normal, and patient otherwise sound.

On February 1st increasing infiltration area around disc, right eye, being more pronounced at temporal side, where it is about two-thirds of a disc diameter in width and somewhat narrower on nasal side of papilla. The appearance of the infiltration presents the popcorn or cumulus cloud effect, and is best seen with a −5 lens. Fine vascularization is noted. The left eye shows indications at temporal border of disc of the same process. The patient has no teeth. A careful examination of the nose and paranasal sinuses is negative. There are, however, small diseased tonsils, exuding pus on pressure. V. R. E., sph. −5 = 20/200 (myopic increase); L. E. = 20/40, no improvement with lenses. On March 1st the exudation about the right disc is bulging out still more in temporo-inferior area. In left eye the exudate now encircles the disc in a band about one-eighth disc diameter in width. Attached to the nasal side of this band is a thin, finely organized, opaque, veil-like substance, presenting edgeways in the vertical plane, about one-half disc diameter in vertical width, and waving in the vitreous. The appearance suggests proliferative retinitis, with a suspicion of fine-spun vascularity. Patient is just recovering from an intercurrent attack of bronchitis. On March 28th, double tonsillectomy.

On May 3d the organized exudate about the right disc is increasing, being now a full disc diameter in width temporally and inferiorly. There is undoubted fine vascularization. From the hyperplastic mass, spreading for two-thirds of the way to the macula, is a whitish haze of exudation, roughly triangular in shape, with base the width of the new growth, beneath the nerve-fiber layer and with characteristic appearance of serous retinitis, evidencing the extension of the exudative process. Organization of this exudation can be seen to be taking place along the course of some of the vessels, contiguous to the already organized mass. Tortuosity of vessels elsewhere in retina. Increased evidences of degenerative changes in macular area, and between that and the disc. Several irregular, bright yellow spots of varying sizes and shapes, and in some are faint appearances of pigment deposits in centers. The left fundus shows marked tortuosity of vessels. The proliferating tissue at nasal side of disc is

appreciably less well defined and seems to be atrophying. V. R. E., with sph. $-7 = 20/200$; L. E. $= 20/30$. Tension (Schiötz tonometer), 18 each eye.

There is an evident increase in the anteroposterior diameter of the right bulb, in marked contrast to the behavior of the left bulb, though the pathologic process in each eye is clearly progressing. This phenomenon, it seems to the writer, can be explained only on the ground of the exertion of traction posteriorly by the process as it extends backward along the nerve-trunk. There is no evidence of lenticular swelling or keratoconus. The lesion at the disc could not possibly be confused with the so-called posterior staphyloma of progressive or high myopia, which, it is now recognized, is not a staphyloma at all, in the real sense of the word. The whole history and picture would confound the suggestion of such confusion, though perhaps the writer's inadequacy of description might incite such a criticism.

Conclusions.—There is a retinal pathologic entity consisting of organized hyperplastic tissue evolving from fibrinous or plastic retinal exudates of indeterminate origin.

These exudates are not of hemorrhagic character, though it would appear that they must necessarily have a vascular pathogenesis, and the tissue into which they are determined would seem to be analogous to granulation or chronic inflammatory tissue.

The clinical phenomena manifested can evidently be divorced from syphilitic, tubercular, or traumatic causation, though unquestionably resembling the lesions sometimes seen consequent upon these origins.

The hyperplastic organizations are characterized by a white or grayish or yellowish or mottled appearance, in form suggesting a piece of popcorn or miniature cumulus clouds, showing a fine vascularity at some period of their development; of no fixed size; usually, it would seem, in one mass, often highly elevated from the retinal level; when in the

active formative stage, usually with well-marked peripheral definitions; with a minimum of pigmentation or none at all; and, in the writer's observation at least, with points of selection for their development either in the papillary or macular area. If in the disc area, the organized exudates probably always extend along the nerve-trunk beneath the sheath. They are, of course, essentially benign.

As the pathologic morphology and clinical appearances of these lesions are evidently similar (though usually distinguishable by location, arrangement, anamnesis, and instruments and methods of precision) to those of retinitis circinata, retinitis proliferans of non-hemorrhagic origin, and certain forms of syphilitic, traumatic, and perhaps tubercular retinitis, it would seem appropriate for clinical study and for the minimizing of a certain obscurity to group all of these, for differentiation from other types, under the descriptive head of hyperplastic exudative retinitis—non-hemorrhagic.

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